Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1653hxp

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	SEP 09	CA/CAplus records now contain indexing from 1907 to the present
NEWS 4	AUG 05	New pricing for EUROPATFULL and PCTFULL effective August 1, 2003
NEWS 5	AUG 13	Field Availability (/FA) field enhanced in BEILSTEIN
NEWS 6	AUG 18	Data available for download as a PDF in RDISCLOSURE
NEWS 7	AUG 18	Simultaneous left and right truncation added to PASCAL
NEWS 8	AUG 18	FROSTI and KOSMET enhanced with Simultaneous Left and Righ Truncation
NEWS 9	AUG 18	Simultaneous left and right truncation added to ANABSTR
NEWS 10	SEP 22	DIPPR file reloaded
NEWS 11	SEP 25	INPADOC: Legal Status data to be reloaded
NEWS 12	SEP 29	DISSABS now available on STN
NEWS 13	OCT 10	PCTFULL: Two new display fields added
NEWS 14	OCT 21	BIOSIS file reloaded and enhanced
NEWS 15	OCT 28	BIOSIS file segment of TOXCENTER reloaded and enhanced
NEWS 16	NOV 24	MSDS-CCOHS file reloaded

NEWS EXPRESS	NOVEMBER 14 CURRENT WINDOWS VERSION IS V6.01c, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003

=> file medline, uspatful, dgene, embase, wpids, biosis, biobusiness

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 11:08:30 ON 01 DEC 2003

FILE 'USPATFULL' ENTERED AT 11:08:30 ON 01 DEC 2003
CA INDEXING COPYRIGHT (C) 2003 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'DGENE' ENTERED AT 11:08:30 ON 01 DEC 2003
COPYRIGHT (C) 2003 THOMSON DERWENT

FILE 'EMBASE' ENTERED AT 11:08:30 ON 01 DEC 2003
COPYRIGHT (C) 2003 Elsevier Inc. All rights reserved.

FILE 'WPIDS' ENTERED AT 11:08:30 ON 01 DEC 2003
COPYRIGHT (C) 2003 THOMSON DERWENT

FILE 'BIOSIS' ENTERED AT 11:08:30 ON 01 DEC 2003
COPYRIGHT (C) 2003 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'BIOBUSINESS' ENTERED AT 11:08:30 ON 01 DEC 2003
COPYRIGHT (C) 2003 Biological Abstracts, Inc. (BIOSIS)

=> s conjugate and mastocyte binding
L1 1 CONJUGATE AND MASTOCYTE BINDING

=> d l1 ti abs ibib tot

L1 ANSWER 1 OF 1 USPATFULL on STN
TI Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof
AB A hybrid protein contains a protein that binds to a receptor of mastocytes and basophils and is endocyted by them. The protein can be IgE; IgE fragment; IgE Fc fragment; antibody against IgE receptor of mastocytes and basophils; fragment of the antibody against the IgE receptor of mastocytes and basophils; antibody against mastocyte specific potassium channel; and mast cell degranulating peptide. The hybrid protein also contains a protease cleaving proteins of the secretion process of the mastocytes and basophils so as to inhibit the secretion process without killing the mastocytes and basophils. The protease can be light chain Clostridium botulinum toxin; proteolytically active fragment of the light chain of a Clostridium botulinum toxin containing an amino acid sequence His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His wherein Xaa is an amino acid; light chain of the tetanus toxin; proteolytically active fragment of the light chain of the tetanus toxin containing His-Asp-Leu-Ile-His-Val-Leu-His; IgA protease of Neisseria gonorrhoeae; and proteolytic domain of the IgA protease of Neisseria gonorrhoeae.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:86306 USPATFULL
TITLE:	Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof
INVENTOR(S):	Bigalke, Hans, Hannover, GERMANY, FEDERAL REPUBLIC OF Frevert, Jurgen, Berlin, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S):	BioteCon Gesellschaft fur biotechnologische Entwicklung und consulting mbH, Berlin, DE, 10589 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059912	A1	20030327
APPLICATION INFO.:	US 2002-64903	A1	20020827 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-700540, filed on 19 Jan 2001, PENDING A 371 of International Ser. No. WO 1999-EP3272, filed on 12 May 1999, UNKNOWN

	NUMBER	DATE

```
                          ------------------ --------
PRIORITY INFORMATION:     DE 1998-19821285   19980513
DOCUMENT TYPE:            Utility
FILE SEGMENT:             APPLICATION
LEGAL REPRESENTATIVE:     GUDRUN E. HUCKETT, LONSSTR. 53, WUPPERTAL, 42289
NUMBER OF CLAIMS:         11
EXEMPLARY CLAIM:          1
LINE COUNT:               576
CAS INDEXING IS AVAILABLE FOR THIS PATENT.


=> d his

     (FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS'
     ENTERED AT 11:08:30 ON 01 DEC 2003
L1             1 S CONJUGATE AND MASTOCYTE BINDING

=> s hybrid protein or conjugate
L2        171149 HYBRID PROTEIN OR CONJUGATE

=> s IgE and IgA protease
L3            21 IGE AND IGA PROTEASE

=> s IgE and tetanus
L4          1711 IGE AND TETANUS

=> s l2 and l3
L5             7 L2 AND L3

=> d l5 ti abs ibibi tot
'IBIBI' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d l5 ti abs ibib tot

L5   ANSWER 1 OF 7  USPATFULL on STN
TI     Hybrid protein for inhibiting the degranulation of
       mastocytes and the use thereof
AB     A hybrid protein contains a protein that binds to a
       receptor of mastocytes and basophils and is endocyted by them. The
       protein can be IgE; IgE fragment; IgE Fc
       fragment; antibody against IgE receptor of mastocytes and
       basophils; fragment of the antibody against the IgE receptor
       of mastocytes and basophils; antibody against mastocyte specific
       potassium channel; and mast cell degranulating peptide. The
       hybrid protein also contains a protease cleaving
       proteins of the secretion process of the mastocytes and basophils so as
       to inhibit the secretion process without killing the mastocytes and
       basophils. The protease can be light chain Clostridium botulinum toxin;
       proteolytically active fragment of the light chain of a Clostridium
       botulinum toxin containing an amino acid sequence His-Xaa-Xaa-Xaa-His-
       Xaa-Xaa-His wherein Xaa is an amino acid; light chain of the tetanus
       toxin; proteolytically active fragment of the light chain of the tetanus
       toxin containing His-Asp-Leu-Ile-His-Val-Leu-His; IgA
       protease of Neisseria gonorrhoeae; and proteolytic domain of the
       IgA protease of Neisseria gonorrhoeae.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
```

ACCESSION NUMBER: 2003:86306 USPATFULL
TITLE: **Hybrid protein** for inhibiting the degranulation of mastocytes and the use thereof
INVENTOR(S): Bigalke, Hans, Hannover, GERMANY, FEDERAL REPUBLIC OF
Frevert, Jurgen, Berlin, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): BioteCon Gesellschaft fur biotechnologische Entwicklung und consulting mbH, Berlin, DE, 10589 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059912	A1	20030327
APPLICATION INFO.:	US 2002-64903	A1	20020827 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-700540, filed on 19 Jan 2001, PENDING A 371 of International Ser. No. WO 1999-EP3272, filed on 12 May 1999, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	DE 1998-19821285	19980513

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GUDRUN E. HUCKETT, LONSSTR. 53, WUPPERTAL, 42289
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
LINE COUNT: 576
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 2 OF 7 USPATFULL on STN
TI Directed evolution of enzymes and antibodies
AB The invention relates to methods of selecting proteins, out of large libraries, having desirable characteristics. Exemplified are methods of expressing enzymes and antibodies on the surface of host cells and selecting for desired activities. These methods have the advantage of speed and ease of operation when compared with current methods. They also provide, without additional cloning, a source of significant quantities of the protein of interest.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:51135 USPATFULL
TITLE: Directed evolution of enzymes and antibodies
INVENTOR(S): Iverson, Brent, Austin, TX, UNITED STATES
Georgiou, George, Austin, TX, UNITED STATES
Chen, Gang, Austin, TX, UNITED STATES
Olsen, Mark J., Austin, TX, UNITED STATES
Daugherty, Patrick S., Austin, TX, UNITED STATES
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003036092	A1	20030220
APPLICATION INFO.:	US 2001-782672	A1	20010212 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1997-847063, filed on 1 May 1997, ABANDONED Continuation-in-part of Ser. No. US 1995-447402, filed on 23 May 1995, GRANTED, Pat. No. US 5866344 Continuation-in-part of Ser. No. US 1994-258543, filed on 10 Jun 1994, ABANDONED Division of Ser. No. US 1991-794731, filed on 15 Nov 1991, GRANTED, Pat. No. US 5348867
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Steven L. Highlander, Esq., FULBRIGHT & JAWORSKI L.L.P., Suite 2400, 600 Congress Avenue, Austin, TX,

78701
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 3955
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 3 OF 7 USPATFULL on STN
TI Methods for producing members of specific binding pairs
AB Methods, recombinant host cells and kits are disclosed-for the production of members of specific binding pairs (sbp), e.g. antibodies, using display on the surface of secreted replicable genetic display packages (rgdps), e.g. filamentous phage. To produce a library of great diversity recombination occurs between first and second vectors comprising nucleic acid encoding first and second polypeptide chains of sbp members respectively, thereby producing recombinant vectors each encoding both a first and a second polypeptide chain component of a sbp member. The recombination may take place in vitro or intracellularly and may be site-specific, e.g. involving use of the loxP sequence and mutants thereof. Recombination may take place after prior screening or selecting for rgdps displaying sbp members which bind complementary sbp member of interest.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:325868 USPATFULL
TITLE: Methods for producing members of specific binding pairs
INVENTOR(S): Griffiths, Andrew David, Cambridge, UNITED KINGDOM
Williams, Samuel Cameron, Cambridge, UNITED KINGDOM
Waterhouse, Peter Michael, Canberra, AUSTRALIA
Nissim, Ahuva, Cambridge, UNITED KINGDOM
Winter, Gregory Paul, Cambridge, UNITED KINGDOM
Johnson, Kevin Stuart, Cambridgeshire, UNITED KINGDOM
Smith, Andrew John Hammond, Cambridge, UNITED KINGDOM
PATENT ASSIGNEE(S): Cambridge Antibody Technology Limited, Cambridgeshire, UNITED KINGDOM (non-U.S. corporation)
Medical Research Council, London, UNITED KINGDOM (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6492160	B1	20021210
APPLICATION INFO.:	US 1998-104337		19980625 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1994-350260, filed on 5 Dec 1994, now patented, Pat. No. US 5962255 Continuation-in-part of Ser. No. US 307619, now patented, Pat. No. US 5733743 Continuation-in-part of Ser. No. US 150002, now patented, Pat. No. US 5871907

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1991-10549	19910515
	GB 1992-6318	19920324

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Ketter, James
LEGAL REPRESENTATIVE: Marshall, Gerstein & Borun.
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 42 Drawing Figure(s); 34 Drawing Page(s)
LINE COUNT: 6137
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 4 OF 7 USPATFULL on STN
TI Compositions and methods for the diagnosis, treatment and prevention of

steroid hormone responsive cancers

AB Compositions and methods that use the body's natural secretory immune system in a new way against steroid hormone responsive tumors of the breast and prostate, as well as other glandular/mucus epithelial tissues such as colon, ovary, endometrium, kidney, bladder, stomach, pancreas and secretory pituitary gland are provided. Also provided are new ways of identifying carcinogenic, or potentially carcinogenic, bacteria in a tissue or body fluid to provide better anti-cancer therapies and preventatives than have been available previously.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2002:12251 USPATFULL			
TITLE:	Compositions and methods for the diagnosis, treatment and prevention of steroid hormone responsive cancers			
INVENTOR(S):	Sirbasku, David A., Houston, TX, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002006630	A1	20020117	
APPLICATION INFO.:	US 2001-852547	A1	20010510	(9)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2000-203314P		20000510	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	CONLEY ROSE & TAYON, P.C., P. O. BOX 3267, HOUSTON, TX, 77253-3267			
NUMBER OF CLAIMS:	65			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	133 Drawing Page(s)			
LINE COUNT:	10394			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 5 OF 7 USPATFULL on STN

TI Recombinant human IGA-J. chain dimer

AB Disclosed are compositions and methods of use that comprise engineered IgA antibodies that, when administered to a host are secreted across the epithelium into the mucosal barriers of the body providing external passive immunotherapy against agents such as viral, bacterial and eukaryotic pathogens. Also disclosed are mini antibodies comprising the minimal transcytosis domains.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2000:61721 USPATFULL			
TITLE:	Recombinant human IGA-J. chain dimer			
INVENTOR(S):	Capra, J. Donald, Dallas, TX, United States Hexham, Jonathan M., Dallas, TX, United States Carayannopoulos, Leon N., St Louis, MO, United States Max, Edward E., Bethesda, MD, United States			
PATENT ASSIGNEE(S):	Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation) The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6063905		20000516	
APPLICATION INFO.:	US 1997-779597		19970107	(8)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Eyler, Yvonne			
LEGAL REPRESENTATIVE:	Arnold, White & Durkee			

NUMBER OF CLAIMS: 102
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 2003
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 6 OF 7 USPATFULL on STN
TI Methods for producing recombinant vectors
AB Methods, recombinant host cells and kits are disclosed for the production of members of specific binding pairs (sbp), e.g. antibodies, using display on the surface of secreted replicable genetic display packages (rgdps), e.g. filamentous phage. To produce a library of great diversity recombination occurs between first and second vectors comprising nucleic acid encoding first and second polypeptide chains of sbp members respectively, thereby producing recombinant vectors each encoding both a first and a second polypeptide chain component of a sbp member. The recombination may take place in vitro or intracellularly and may be site-specific, e.g. involving use of the loxP sequence and mutants thereof. Recombination may take place after prior screening or selecting for rgdps displaying sbp members which bind complementary sbp member of interest.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 1999:121158 USPATFULL
TITLE: Methods for producing recombinant vectors
INVENTOR(S): Griffiths, Andrew David, Cambridge, United Kingdom
Williams, Samuel Cameron, Cambridge, United Kingdom
Waterhouse, Peter Michael, Canberra, Australia
Nissim, Ahuva, Cambridge, United Kingdom
Winter, Gregory Paul, Cambridge, United Kingdom
Johnson, Kevin Stuart, Cambridgeshire, United Kingdom
Smith, Andrew John Hammond, Cambridge, United Kingdom
PATENT ASSIGNEE(S): Cambridge Antibody Technology Limited, Cambridgeshire, United Kingdom (non-U.S. corporation)
Medical Research Council, London, United Kingdom (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5962255		19991005
APPLICATION INFO.:	US 1994-350260		19941205 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-307619, filed on 16 Sep 1994 which is a continuation-in-part of Ser. No. US 1994-150002, filed on 31 Mar 1994

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1992-6318	19920324

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Ketter, James
ASSISTANT EXAMINER: Wai, Thanda
LEGAL REPRESENTATIVE: Marshall, O'Toole, Gerstein, Murray & Borun
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 42 Drawing Figure(s); 34 Drawing Page(s)
LINE COUNT: 7715
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 7 OF 7 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
TI New **hybrid protein** useful for inhibiting mast cell degranulation and treating allergic reactions.
AN 2000-072332 [06] WPIDS
AB WO 9958571 A UPAB: 20000203

NOVELTY - A protein which binds to, or is absorbed by, mast cells or basophils is combined with a known protease (which cleaves proteins of the secretory apparatus of such cells) in a **hybrid protein** which is useful for inhibiting mast cell degranulation and treating allergic reactions.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for: (A) **hybrid protein** comprising: (a) a known protein which binds to (or is absorbed by) mast cells and/or basophils, in a known manner; and (b) a known protease which cleaves one or more proteins of the secretory apparatus of the mast cells or basophils. (B) **hybrid protein** comprising: (a) a protein which binds to (or is absorbed by) mast cells or basophils; and (b) a protease (especially a known protease) which cleaves one or more proteins of the secretory apparatus of the mast cells or basophils. Component (a) is selected from (i) **IgE**, (ii) **IgE** fragments (especially an **IgE**-Fc fragment), (iii) antibodies against **IgE** receptors of mast cells and/or basophils, (iv) fragments of antibodies against **IgE** receptors of mast cells and/or basophils (especially an Fab fragment), (v) antibodies against the mast cell-specific potassium channel, and (vi) inactive (though binding) MCD peptide. (C) **hybrid protein** comprising: (a) a protein (especially a known protein) which binds to (or is absorbed by) mast cells and/or basophils; and (b) a protease which cleaves one or more proteins of the secretion apparatus of the mast cells or basophils. The protease is selected from (i) the light chain of a Clostridum botulinum toxin (especially type A, B, Cl, D, E, F or G), (ii) the light chain of Tetanus toxin, (iii) catalytically active fragments of the light chains described in (i) or (ii), (iv) **IgA protease** from Neisseria gonorrhea or (v) catalytic domains of **IgA protease** from Neisseria gonorrhea.

ACTIVITY - Antiallergic.

USE - The hybrid proteins inhibit mast cell degranulation, and may be used in treatment or prevention of allergic reactions.

Dwg.0/0

ACCESSION NUMBER: 2000-072332 [06] WPIDS
DOC. NO. CPI: C2000-020614
TITLE: New **hybrid protein** useful for inhibiting mast cell degranulation and treating allergic reactions.
DERWENT CLASS: B04 D16 J04
INVENTOR(S): BIGALKE, H; FREVERT, J
PATENT ASSIGNEE(S): (BIOT-N) BIOTECON-GES BIOTECHNOLOGISCHE; (BIET-N) BIETECON GES BIOTECHNOLOGISCHE ENTWICKLU; (BIOT-N) BIOTECON-GES BIOTECHNOLOGISCHE ENTWICKLU
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9958571	A2	19991118	(200006)*	GE	22
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9942605	A	19991129	(200018)		
BR 9910359	A	20010109	(200106)		
NO 2000005637	A	20001108	(200108)		
EP 1084146	A2	20010321	(200117)	GE	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE					
CZ 2000004161	A3	20010411	(200130)		
CN 1300295	A	20010620	(200159)		
KR 2001042825	A	20010525	(200168)		
HU 2001003601	A2	20020128	(200222)		

JP 2002514659 W 20020521 (200236) 22
EP 1084146 B1 20021113 (200282) GE
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE
DE 59903410 G 20021219 (200302)
AU 755513 B 20021212 (200305)
US 2003059912 A1 20030327 (200325)
ES 2187200 T3 20030516 (200337)

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9958571	A2		WO 1999-EP3272	19990512
AU 9942605	A		AU 1999-42605	19990512
BR 9910359	A		BR 1999-10359	19990512
			WO 1999-EP3272	19990512
NO 2000005637	A		WO 1999-EP3272	19990512
			NO 2000-5637	20001108
EP 1084146	A2		EP 1999-950347	19990512
			WO 1999-EP3272	19990512
CZ 2000004161	A3		WO 1999-EP3272	19990512
			CZ 2000-4161	19990512
CN 1300295	A		CN 1999-806061	19990512
KR 2001042825	A		KR 2000-711584	20001018
HU 2001003601	A2		WO 1999-EP3272	19990512
			HU 2001-3601	19990512
JP 2002514659	W		WO 1999-EP3272	19990512
			JP 2000-548373	19990512
EP 1084146	B1		EP 1999-950347	19990512
			WO 1999-EP3272	19990512
DE 59903410	G		DE 1999-503410	19990512
			EP 1999-950347	19990512
			WO 1999-EP3272	19990512
AU 755513	B		AU 1999-42605	19990512
US 2003059912	A1	CIP of	WO 1999-EP3272	19990512
		CIP of	US 2001-700540	20010119
			US 2002-64903	20020827
ES 2187200	T3		EP 1999-950347	19990512

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9942605	A	Based on	WO 9958571
BR 9910359	A	Based on	WO 9958571
EP 1084146	A2	Based on	WO 9958571
CZ 2000004161	A3	Based on	WO 9958571
HU 2001003601	A2	Based on	WO 9958571
JP 2002514659	W	Based on	WO 9958571
EP 1084146	B1	Based on	WO 9958571
DE 59903410	G	Based on	EP 1084146
		Based on	WO 9958571
AU 755513	B	Previous Publ.	AU 9942605
		Based on	WO 9958571
ES 2187200	T3	Based on	EP 1084146

PRIORITY APPLN. INFO: DE 1998-19821285 19980513

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS'

```
     ENTERED AT 11:08:30 ON 01 DEC 2003
L1             1 S CONJUGATE AND MASTOCYTE BINDING
L2        171149 S HYBRID PROTEIN OR CONJUGATE
L3            21 S IGE AND IGA PROTEASE
L4          1711 S IGE AND TETANUS
L5             7 S L2 AND L3

=> s l4 and mastocyte inactivation
L6             0 L4 AND MASTOCYTE INACTIVATION

=> s l4 and degranulation inhibition
L7             0 L4 AND DEGRANULATION INHIBITION

=> s mast cell degranulation and inhibition
L8          1322 MAST CELL DEGRANULATION AND INHIBITION

=> s allergy and treatement
L9            38 ALLERGY AND TREATEMENT

=> s l9 and l8
L10            0 L9 AND L8

=> s l8 and allergic response
L11          107 L8 AND ALLERGIC RESPONSE

=> s l11 and IgE
L12           88 L11 AND IGE

=> s l12 and tetanus toxin
L13            2 L12 AND TETANUS TOXIN

=> d l13 ti abs ibib tot

L13  ANSWER 1 OF 2  USPATFULL on STN
TI     Bi-directionally cloned random cDNA expression vector libraries,
       compositions and methods of use
AB     The present invention provides random cDNA expression vector libraries,
       comprising expression vectors which comprise random cDNAs positioned in
       sense and antisense orientation, which are useful for the delivery and
       expression of a combination of genetic effector types to host cells.
       Methods for producing these libraries through bi-directional cloning of
       random cDNAs are also provided. Also provided herein are methods of
       using these libraries to screen for agents capable of modulating cell
       phenotype in desirable ways.

ACCESSION NUMBER:         2003:300312  USPATFULL
TITLE:                    Bi-directionally cloned random cDNA expression vector
                          libraries, compositions and methods of use
INVENTOR(S):              Lorens, James, Portola Valley, CA, UNITED STATES
                          Bogenberger, Jakob M., San Francisco, CA, UNITED STATES

                               NUMBER         KIND    DATE
                          ---------------- ------ --------
PATENT INFORMATION:       US 2003211535      A1   20031113
APPLICATION INFO.:        US 2002-142648     A1   20020508  (10)
DOCUMENT TYPE:            Utility
FILE SEGMENT:             APPLICATION
LEGAL REPRESENTATIVE:     BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD,
                          SUITE 200, MENLO PARK, CA, 94025
NUMBER OF CLAIMS:         20
EXEMPLARY CLAIM:          1
NUMBER OF DRAWINGS:       5 Drawing Page(s)
LINE COUNT:               3910
```

L13 ANSWER 2 OF 2 USPATFULL on STN
TI Directionally cloned random cDNA expression vector libraries, compositions and methods of use
AB The present invention provides random cDNA expression vector libraries, comprising expression vectors which comprise random cDNAs positioned in sense orientation. Also provided are random cDNA expression vector libraries, comprising expression vectors which comprise random cDNAs positioned in antisense orientation. Methods for producing these libraries through directional cloning of random cDNAs are also provided. Also provided herein are methods of using these libraries to screen for agents capable of modulating cell phenotype in desirable ways.

ACCESSION NUMBER:	2003:300239 USPATFULL			
TITLE:	Directionally cloned random cDNA expression vector libraries, compositions and methods of use			
INVENTOR(S):	Shen, Mary, Newark, CA, UNITED STATES Yu, Simon, Newark, CA, UNITED STATES Wu, Xian, Redwood City, CA, UNITED STATES Payan, Donald, Hillsborough, CA, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003211462	A1	20031113	
APPLICATION INFO.:	US 2002-142662	A1	20020508	(10)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025			
NUMBER OF CLAIMS:	26			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	4 Drawing Page(s)			
LINE COUNT:	3873			

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS'
ENTERED AT 11:08:30 ON 01 DEC 2003

L1	1	S	CONJUGATE AND MASTOCYTE BINDING
L2	171149	S	HYBRID PROTEIN OR CONJUGATE
L3	21	S	IGE AND IGA PROTEASE
L4	1711	S	IGE AND TETANUS
L5	7	S	L2 AND L3
L6	0	S	L4 AND MASTOCYTE INACTIVATION
L7	0	S	L4 AND DEGRANULATION INHIBITION
L8	1322	S	MAST CELL DEGRANULATION AND INHIBITION
L9	38	S	ALLERGY AND TREATEMENT
L10	0	S	L9 AND L8
L11	107	S	L8 AND ALLERGIC RESPONSE
L12	88	S	L11 AND IGE
L13	2	S	L12 AND TETANUS TOXIN

=> d l12 ti abs ibib 1-10

L12 ANSWER 1 OF 88 MEDLINE on STN
TI Shini-san inhibits mast cell-dependent immediate-type allergic reactions.
AB Shini-San has been used for treatment of allergic disease in Korea. However, its effect in experimental models remains unknown. The mast cell plays a pivotal role in initiating **allergic response** by secreting intracytoplasmic granular mediators such as histamine. The present report describes an inhibitory effect of Shini-San on mast cell-mediated immediate-type allergic reactions. Topical application of

compound 48/80 can induce an ear swelling response in normal (WBB6F1(-)+/+) mice but not in congenic mast cell-deficient WBB6F1-W/WV mice. Shini-San inhibited concentration-dependent mast cell-dependent ear swelling response induced by compound 48/80 in normal mice. Shini-San inhibited concentration-dependent passive cutaneous anaphylaxis induced by anti-dinitrophenyl (DNP) immunoglobulin E (**IgE**) in rats by topical application. Shini-San also inhibited in concentration-dependent fashion the histamine release from the rat peritoneal mast cells by compound 48/80 or anti-DNP **IgE**. Moreover, Shini-San had a significant inhibitory effect on compound 48/80-induced systemic anaphylactic reaction. These results indicate that Shini-San inhibits immediate type allergic reactions by **inhibition** of **mast cell degranulation** in vivo and in vitro.

ACCESSION NUMBER: 2000060432 MEDLINE
DOCUMENT NUMBER: 20060432 PubMed ID: 10592847
TITLE: Shini-san inhibits mast cell-dependent immediate-type allergic reactions.
AUTHOR: Kim H M; Lee Y H; Chae H J; Kim H R; Baek S H; Lim K S; Hwang C Y
CORPORATE SOURCE: Department of Oriental Pharmacy, College of Pharmacy, Wonkwang University, Iksan, Chonbuk, South Korea.
SOURCE: AMERICAN JOURNAL OF CHINESE MEDICINE, (1999) 27 (3-4) 377-86.
Journal code: 7901431. ISSN: 0192-415X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000209
Last Updated on STN: 20000209
Entered Medline: 20000203

L12 ANSWER 2 OF 88 MEDLINE on STN
TI Magnoliae flos inhibits mast cell-dependent immediate-type allergic reactions.
AB The mast cell plays a pivotal role in initiating **allergic response** by secreting intracytoplasmic granular mediators such as histamine. Magnoliae flos has been used for the treatment of allergic disease in Korea. However, its effect in experimental models remains unknown. The present report describes an inhibitory effect of Magnoliae flos on mast cell-mediated immediate-type allergic reactions. Topical application of compound 48/80 can induce an ear swelling response in normal (WBB6F1-+/+) mice but not in the congenic mast cell-deficient WBB6F1-W/Wv mice. Magnoliae flos inhibited concentration-dependently mast cell-dependent ear swelling response induced by compound 48/80 by topical application. Magnoliae flos inhibited concentration-dependently passive cutaneous anaphylaxis induced by anti-dinitrophenyl (DNP) **IgE** in rats by topical application. Magnoliae flos also inhibited concentration-dependently the histamine release from the rat peritoneal mast cells by compound 48/80 or anti-DNP **IgE**. Moreover, Magnoliae flos had a significant inhibitory effect on compound 48/80-induced systemic anaphylactic reaction. These results indicate that Magnoliae flos inhibits immediate-type allergic reactions by **inhibition** of **mast cell degranulation** in vivo and in vitro.

ACCESSION NUMBER: 1999174063 MEDLINE
DOCUMENT NUMBER: 99174063 PubMed ID: 10072701
TITLE: Magnoliae flos inhibits mast cell-dependent immediate-type allergic reactions.
AUTHOR: Kim H M; Yi J M; Lim K S
CORPORATE SOURCE: College of Pharmacy, Wonkwang University, Iksan, Chonbuk, 570-749, South Korea.

SOURCE: PHARMACOLOGICAL RESEARCH, (1999 Feb) 39 (2) 107-11.
Journal code: 8907422. ISSN: 1043-6618.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 19990511
Last Updated on STN: 19990511
Entered Medline: 19990427

L12 ANSWER 3 OF 88 MEDLINE on STN

TI A sensitive colorimetric assay for the release of tryptase from human lung mast cells in vitro.

AB Studies of human lung mast cells have usually focused on histamine release, although the enzymes stored in the granules may also contribute to the pathophysiology of the **allergic response**. We have used a simple colorimetric assay for tryptase to follow the release of proteolytic enzymes from human lung mast cells in vitro. Either human lung mast cell supernatants or authentic mast cell tryptase were mixed with benzoyl-DL-arginine-p-nitroaniline and incubated for up to 72 h at 37 degrees C. The appearance of nitroaniline was then measured at 410 nm in an ELISA plate reader. Cells were sonicated in H2O to measure total tryptase and histamine. Human lung mast cells contained the equivalent of 11.2 +/- 0.7 pg tryptase per cell and 3.2 +/- 0.3 pg of histamine. The amount of tryptase measured colorimetrically correlated with the level of tryptase measured by radioimmunoassay (Pharmacia), $r = 0.92$, $P < 0.01$. The **inhibition** profile of the proteolytic enzyme measured by the cleavage of BAPNA, was found to be identical to that of authentic lung mast cell tryptase. Over 90% of the maximum tryptase release was complete within 15 min whilst histamine release occurred within 5 min. In cells stimulated with 10 micrograms/ml anti-**IgE** we found a strong correlation between the release of tryptase and histamine, $r = 0.95$, $P < 0.005$. Finally, investigations with various pharmacological agents have supported our initial hypothesis that tryptase would mimic histamine release and provide an alternative marker for mast cell activation. In summary, we have utilised a simple enzymic assay as an indicator of human lung **mast cell degranulation**. In washed lung mast cells this assay appears be specific for granule tryptase and release of this activity into the supernatants of challenged cells correlates well with the presence of histamine. This assay offers several advantages over current methods of measuring mediator release from human lung mast cells in vitro and should provide an inexpensive and sensitive technique for following **mast cell degranulation**.

ACCESSION NUMBER: 94044840 MEDLINE
DOCUMENT NUMBER: 94044840 PubMed ID: 7693824
TITLE: A sensitive colorimetric assay for the release of tryptase from human lung mast cells in vitro.
AUTHOR: Lavens S E; Proud D; Warner J A
CORPORATE SOURCE: Department of Physiology and Pharmacology, University of Southampton, Bassett Crescent East, UK.
CONTRACT NUMBER: HL 32272 (NHLBI)
SOURCE: JOURNAL OF IMMUNOLOGICAL METHODS, (1993 Nov 5) 166 (1) 93-102.
Journal code: 1305440. ISSN: 0022-1759.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 20000303
Entered Medline: 19931214

L12 ANSWER 4 OF 88 USPATFULL on STN
TI Bi-directionally cloned random cDNA expression vector libraries, compositions and methods of use
AB The present invention provides random cDNA expression vector libraries, comprising expression vectors which comprise random cDNAs positioned in sense and antisense orientation, which are useful for the delivery and expression of a combination of genetic effector types to host cells. Methods for producing these libraries through bi-directional cloning of random cDNAs are also provided. Also provided herein are methods of using these libraries to screen for agents capable of modulating cell phenotype in desirable ways.

ACCESSION NUMBER: 2003:300312 USPATFULL
TITLE: Bi-directionally cloned random cDNA expression vector libraries, compositions and methods of use
INVENTOR(S): Lorens, James, Portola Valley, CA, UNITED STATES
Bogenberger, Jakob M., San Francisco, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003211535	A1	20031113	
APPLICATION INFO.:	US 2002-142648	A1	20020508	(10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 3910

L12 ANSWER 5 OF 88 USPATFULL on STN
TI Directionally cloned random cDNA expression vector libraries, compositions and methods of use
AB The present invention provides random cDNA expression vector libraries, comprising expression vectors which comprise random cDNAs positioned in sense orientation. Also provided are random cDNA expression vector libraries, comprising expression vectors which comprise random cDNAs positioned in antisense orientation. Methods for producing these libraries through directional cloning of random cDNAs are also provided. Also provided herein are methods of using these libraries to screen for agents capable of modulating cell phenotype in desirable ways.

ACCESSION NUMBER: 2003:300239 USPATFULL
TITLE: Directionally cloned random cDNA expression vector libraries, compositions and methods of use
INVENTOR(S): Shen, Mary, Newark, CA, UNITED STATES
Yu, Simon, Newark, CA, UNITED STATES
Wu, Xian, Redwood City, CA, UNITED STATES
Payan, Donald, Hillsborough, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003211462	A1	20031113	
APPLICATION INFO.:	US 2002-142662	A1	20020508	(10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 3873

L12 ANSWER 6 OF 88 USPATFULL on STN

TI IMPROVED HERBAL COMPOSITION HAVING ANTIALLERGIC PROPERTIES AND A PROCESS FOR THE PREPARATION THEREOF

AB The present invention relating to a herbal antiallergic composition which comprises a synergistic mixture of extracts from the fruits of Terminalia chebula, bark of Albizia lebbeck, Terminalia bellerica and Emblica officinalis. The present invention also contains the fruits of Piper longum. Piper nigrum and of rhizomes of Zingiber officinale and thoroughly mixed to get the final composition which has potent antiallergic activity. The invention also relates to a process for the preparation of such composition. The composition is particularly useful for the treatment of allergic conditions.

ACCESSION NUMBER: 2003:276427 USPATFULL
TITLE: IMPROVED HERBAL COMPOSITION HAVING ANTIALLERGIC PROPERTIES AND A PROCESS FOR THE PREPARATION THEREOF
INVENTOR(S): Agarwal, Ravindra Kumar, Bangalore, INDIA
Agarwal, Anurag, Bangalore, INDIA

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003194452	A1	20031016	
APPLICATION INFO.:	US 2001-19389	A1	20011228	(10)
	WO 2001-IN21		20010223	

	NUMBER	DATE
PRIORITY INFORMATION:	IN 2000-1582000	20000228

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MERCHANT & GOULD PC, P.O. BOX 2903, MINNEAPOLIS, MN, 55402-0903
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 1446

L12 ANSWER 7 OF 88 USPATFULL on STN

TI Compounds that modulate processes associated with **IgE** production and methods and kits for identifying and using the same

AB The present provides compounds capable of modulating IL-4 receptor-mediated **IgE** production, as well as IL-4 induced processes associated therewith, methods and kits for identifying such compounds that utilize a retinoid X receptor as a surrogate analyte and methods of using the compounds in a variety of in vitro, in vitro and ex vivo contexts.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:244329 USPATFULL
TITLE: Compounds that modulate processes associated with **IgE** production and methods and kits for identifying and using the same
INVENTOR(S): Kinsella, Todd M., Fayetteville, NC, UNITED STATES
Masuda, Esteban, Menlo Park, CA, UNITED STATES
Bennett, Mark K., Moraga, CA, UNITED STATES
Warner, Justin E., San Francisco, CA, UNITED STATES
Anderson, David C., San Bruno, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003170738	A1	20030911	
APPLICATION INFO.:	US 2002-98243	A1	20020315	(10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2002-95659, filed on 8 Mar 2002, PENDING			

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: COOLEY GODWARD, LLP, 3000 EL CAMINO REAL, 5 PALO ALTO SQUARE, PALO ALTO, CA, 94306
NUMBER OF CLAIMS: 44
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 3063
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 8 OF 88 USPATFULL on STN
TI Cyclic peptides and analogs useful to treat allergies
AB The present provides cyclic compounds capable of modulating **IgE** production, as well as IL-4 induced processes associated therewith, and methods of using the cyclic compounds in a variety of in vitro and in vitro contexts.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:237981 USPATFULL
TITLE: Cyclic peptides and analogs useful to treat allergies
INVENTOR(S): Kinsella, Todd, Fayetteville, NC, UNITED STATES
Ohashi, Cara, San Francisco, CA, UNITED STATES
Anderson, Dave, San Bruno, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003166138	A1	20030904	
APPLICATION INFO.:	US 2002-197927	A1	20020716	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2002-358827P	20020221	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: DORSEY & WHITNEY LLP, INTELLECTUAL PROPERTY DEPARTMENT, 4 EMBARCADERO CENTER, SUITE 3400, SAN FRANCISCO, CA, 94111
NUMBER OF CLAIMS: 52
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Page(s)
LINE COUNT: 3117
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 9 OF 88 USPATFULL on STN
TI Fcepsilon-PE chimeric protein for targeted treatment of allergy responses a method for its production and pharmaceutical compositions containing the same
AB The present invention generally relates to a new approach for thetherapy of allergic responses, based on targeted elimination of cells expressing the Fc.epsilon.RI receptor by a chimeric cytotoxin FC.sub.2'-3-PE.sub.40. A sequence encoding amino acids 301-437 of the Fc region of the mouse **IgE** molecule was genetically fused to PE.sub.40'--a truncated form of PE lacking the cell binding domain. The chimeric protein, produced in E. coli, specifically and efficiently kills mouse mast cell lines expressing the Fc.epsilon.RI receptor, as well as primary mast cells derived from bone marrow. The present invention provides a chimeric protein for targeted elimination of Fc.epsilon.RI expressing cells especially useful for the therapy of allergic responses. The said chimeric protein is comprised of a cell targeting moiety for Fc.epsilon.RI expressing cells and a cell killing moiety. The preferred killing moiety is the bacterial toxin Pseudomonas exotoxin (PE). This Pseudomonas exotoxin is a product of Pseudomonas aeruginosa. The present invention also relates to a method for the preparation of said protein. This chimeric protein is prepared by genetically fusing

the Fc region of the mouse **IgE** molecule to PE.sub.40, a truncated form of PE lacking the cell binding domain. The present invention also provides pharmaceutical compositions, for the treatment of allergic diseases and for the treatment of hyperplasias and malignancies, comprising as an active ingredient the above mentioned chimeric protein and a conventional adjuvant product.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:226577 USPATFULL		
TITLE:	Fcepsilon-PE chimeric protein for targeted treatment of allergy responses a method for its production and pharmaceutical compositions containing the same		
INVENTOR(S):	Fishman, Ala, Haita, ISRAEL Yarkoni, Shai, Kfar-Saba, ISRAEL Lorberboumgalski, Haya, Jerusalem, ISRAEL		
PATENT ASSIGNEE(S):	YISSUM RESEARCH DEVELOPMENT COMPANY OF THE HEBREW UNIVERSITY OF JERUSALEM (non-U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003158390	A1	20030821
APPLICATION INFO.:	US 2002-96840	A1	20020314 (10)
RELATED APPLN. INFO.:	Division of Ser. No. US 1998-91645, filed on 18 Jun 1998, PENDING		
	NUMBER	DATE	
PRIORITY INFORMATION:	WO 1996-IL181	19961218	
	IL 1995-116436	19951218	
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	LOWE HAUPTMAN GILMAN AND BERNER, LLP, 1700 DIAGONAL ROAD, SUITE 300 /310, ALEXANDRIA, VA, 22314		
NUMBER OF CLAIMS:	11		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	17 Drawing Page(s)		
LINE COUNT:	1038		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 10 OF 88 USPATFULL on STN

TI HERBAL REMEDIES FOR TREATING ALLERGIES AND ASTHMA

AB The present invention provides herbal compositions that can prevent or reduce the severity, intensity, or duration of allergic and/or asthmatic symptoms and/or can prevent or delay the development of an allergic or asthmatic response to an antigen. The compositions may optionally include one or more adjuvants, cytokines, encapsulating materials, or pharmaceutical carriers or excipients, and may be administered prior to, during, or after the development of allergic or asthmatic symptoms in sensitized individuals. Alternatively or additionally, the compositions may be administered prior to sensitization to a particular antigen; preferably substantially concurrently with exposure to the antigen.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:225315 USPATFULL		
TITLE:	HERBAL REMEDIES FOR TREATING ALLERGIES AND ASTHMA		
INVENTOR(S):	Li, Xiu-Min, Mamaroneck, NY, UNITED STATES Sampson, Hugh A., Larchmont, NY, UNITED STATES		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003157126	A1	20030821
	US 6630176	B2	20031007
APPLICATION INFO.:	US 2001-800815	A1	20010307 (9)

```
                              NUMBER         DATE
                     ------------------ --------
PRIORITY INFORMATION:   US 2000-187614P    20000307 (60)
DOCUMENT TYPE:          Utility
FILE SEGMENT:           APPLICATION
LEGAL REPRESENTATIVE:   DORSEY & WHITNEY LLP, INTELLECTUAL PROPERTY DEPARTMENT,
                        250 PARK AVENUE, NEW YORK, NY, 10177
NUMBER OF CLAIMS:       51
EXEMPLARY CLAIM:        1
NUMBER OF DRAWINGS:     27 Drawing Page(s)
LINE COUNT:             2458
CAS INDEXING IS AVAILABLE FOR THIS PATENT.


=> d his

     (FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS'
     ENTERED AT 11:08:30 ON 01 DEC 2003
L1             1 S CONJUGATE AND MASTOCYTE BINDING
L2        171149 S HYBRID PROTEIN OR CONJUGATE
L3            21 S IGE AND IGA PROTEASE
L4          1711 S IGE AND TETANUS
L5             7 S L2 AND L3
L6             0 S L4 AND MASTOCYTE INACTIVATION
L7             0 S L4 AND DEGRANULATION INHIBITION
L8          1322 S MAST CELL DEGRANULATION AND INHIBITION
L9            38 S ALLERGY AND TREATEMENT
L10            0 S L9 AND L8
L11          107 S L8 AND ALLERGIC RESPONSE
L12           88 S L11 AND IGE
L13            2 S L12 AND TETANUS TOXIN

=> s light chain clostridium botulinum toxin
L14            1 LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN

=> d l14 ti abs ibib tot

L14  ANSWER 1 OF 1  USPATFULL on STN
TI     Hybrid protein for inhibiting the degranulation of mastocytes and the
       use thereof
AB     A hybrid protein contains a protein that binds to a receptor of
       mastocytes and basophils and is endocyted by them. The protein can be
       IgE; IgE fragment; IgE Fc fragment; antibody against IgE receptor of
       mastocytes and basophils; fragment of the antibody against the IgE
       receptor of mastocytes and basophils; antibody against mastocyte
       specific potassium channel; and mast cell degranulating peptide. The
       hybrid protein also contains a protease cleaving proteins of the
       secretion process of the mastocytes and basophils so as to inhibit the
       secretion process without killing the mastocytes and basophils. The
       protease can be light chain Clostridium
       botulinum toxin; proteolytically active fragment of
       the light chain of a Clostridium botulinum toxin containing an amino
       acid sequence His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His wherein Xaa is an amino
       acid; light chain of the tetanus toxin; proteolytically active fragment
       of the light chain of the tetanus toxin containing His-Asp-Leu-Ile-His-
       Val-Leu-His; IgA protease of Neisseria gonorrhoeae; and proteolytic
       domain of the IgA protease of Neisseria gonorrhoeae.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:       2003:86306  USPATFULL
TITLE:                  Hybrid protein for inhibiting the degranulation of
                        mastocytes and the use thereof
```

```
INVENTOR(S):            Bigalke, Hans, Hannover, GERMANY, FEDERAL REPUBLIC OF
                        Frevert, Jurgen, Berlin, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S):     BioteCon Gesellschaft fur biotechnologische Entwicklung
                        und consulting mbH, Berlin, DE, 10589 (non-U.S.
                        corporation)

                             NUMBER        KIND    DATE
                        ---------------- ------ --------
PATENT INFORMATION:     US 2003059912      A1   20030327
APPLICATION INFO.:      US 2002-64903      A1   20020827  (10)
RELATED APPLN. INFO.:   Continuation-in-part of Ser. No. US 2001-700540, filed
                        on 19 Jan 2001, PENDING A 371 of International Ser. No.
                        WO 1999-EP3272, filed on 12 May 1999, UNKNOWN

                               NUMBER         DATE
                        ------------------ --------
PRIORITY INFORMATION:   DE 1998-19821285   19980513
DOCUMENT TYPE:          Utility
FILE SEGMENT:           APPLICATION
LEGAL REPRESENTATIVE:   GUDRUN E. HUCKETT, LONSSTR. 53, WUPPERTAL, 42289
NUMBER OF CLAIMS:       11
EXEMPLARY CLAIM:        1
LINE COUNT:             576
CAS INDEXING IS AVAILABLE FOR THIS PATENT.


=> s light chain tetanus toxin
L15           0 LIGHT CHAIN TETANUS TOXIN

=> s IgA protease Neisseria gonorrheae
L16           0 IGA PROTEASE NEISSERIA GONORRHEAE

=> s tetanus and IgE
L17        1711 TETANUS AND IGE

=> d his

     (FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS'
     ENTERED AT 11:08:30 ON 01 DEC 2003
L1             1 S CONJUGATE AND MASTOCYTE BINDING
L2        171149 S HYBRID PROTEIN OR CONJUGATE
L3            21 S IGE AND IGA PROTEASE
L4          1711 S IGE AND TETANUS
L5             7 S L2 AND L3
L6             0 S L4 AND MASTOCYTE INACTIVATION
L7             0 S L4 AND DEGRANULATION INHIBITION
L8          1322 S MAST CELL DEGRANULATION AND INHIBITION
L9            38 S ALLERGY AND TREATEMENT
L10            0 S L9 AND L8
L11          107 S L8 AND ALLERGIC RESPONSE
L12           88 S L11 AND IGE
L13            2 S L12 AND TETANUS TOXIN
L14            1 S LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN
L15            0 S LIGHT CHAIN TETANUS TOXIN
L16            0 S IGA PROTEASE NEISSERIA GONORRHEAE
L17         1711 S TETANUS AND IGE

=> l8 and l17
L8 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).
```

```
=> s l8 and l17
L18         12 L8 AND L17

=> d l18 ti abs ibib tot

L18  ANSWER 1 OF 12  USPATFULL on STN
TI     Compositions and methods for screening and identifying anti-HCV agents
AB     The field of the invention is methods for screening for effector
       peptides, polypeptides and fragments thereof and RNA molecules selected
       inside living cells that have anti-HCV activity.

ACCESSION NUMBER:        2003:312126  USPATFULL
TITLE:                   Compositions and methods for screening and identifying
                         anti-HCV agents
INVENTOR(S):             Lu, Henry H., Foster City, CA, UNITED STATES
                         Huang, Peiyong, Sunnyvale, CA, UNITED STATES
                         Kinsella, Todd, Joliet, IL, UNITED STATES
                         Martinez, Anthony, San Francisco, CA, UNITED STATES

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2003219723      A1   20031127
APPLICATION INFO.:       US 2002-152163     A1   20020520  (10)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    DORSEY & WHITNEY LLP, INTELLECTUAL PROPERTY DEPARTMENT,
                         4 EMBARCADERO CENTER, SUITE 3400, SAN FRANCISCO, CA,
                         94111
NUMBER OF CLAIMS:        28
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      10 Drawing Page(s)
LINE COUNT:              3162

L18  ANSWER 2 OF 12  USPATFULL on STN
TI     Bi-directionally cloned random cDNA expression vector libraries,
       compositions and methods of use
AB     The present invention provides random cDNA expression vector libraries,
       comprising expression vectors which comprise random cDNAs positioned in
       sense and antisense orientation, which are useful for the delivery and
       expression of a combination of genetic effector types to host cells.
       Methods for producing these libraries through bi-directional cloning of
       random cDNAs are also provided. Also provided herein are methods of
       using these libraries to screen for agents capable of modulating cell
       phenotype in desirable ways.

ACCESSION NUMBER:        2003:300312  USPATFULL
TITLE:                   Bi-directionally cloned random cDNA expression vector
                         libraries, compositions and methods of use
INVENTOR(S):             Lorens, James, Portola Valley, CA, UNITED STATES
                         Bogenberger, Jakob M., San Francisco, CA, UNITED STATES

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2003211535      A1   20031113
APPLICATION INFO.:       US 2002-142648     A1   20020508  (10)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD,
                         SUITE 200, MENLO PARK, CA, 94025
NUMBER OF CLAIMS:        20
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      5 Drawing Page(s)
LINE COUNT:              3910
```

L18 ANSWER 3 OF 12 USPATFULL on STN

TI Directionally cloned random cDNA expression vector libraries, compositions and methods of use

AB The present invention provides random cDNA expression vector libraries, comprising expression vectors which comprise random cDNAs positioned in sense orientation. Also provided are random cDNA expression vector libraries, comprising expression vectors which comprise random cDNAs positioned in antisense orientation. Methods for producing these libraries through directional cloning of random cDNAs are also provided. Also provided herein are methods of using these libraries to screen for agents capable of modulating cell phenotype in desirable ways.

ACCESSION NUMBER:	2003:300239 USPATFULL
TITLE:	Directionally cloned random cDNA expression vector libraries, compositions and methods of use
INVENTOR(S):	Shen, Mary, Newark, CA, UNITED STATES Yu, Simon, Newark, CA, UNITED STATES Wu, Xian, Redwood City, CA, UNITED STATES Payan, Donald, Hillsborough, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003211462	A1	20031113	
APPLICATION INFO.:	US 2002-142662	A1	20020508	(10)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025			
NUMBER OF CLAIMS:	26			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	4 Drawing Page(s)			
LINE COUNT:	3873			

L18 ANSWER 4 OF 12 USPATFULL on STN

TI Bio-energy muscle relaxants

AB Human muscle tissues involve striated and smooth muscles. Each muscle tissue possesses its own special function. Differences of physiology functions among the muscle tissues are mainly determined by their various initiation and signal transmission systems, defined as the pre-muscle molecular motor mechanism, or initiating and regulating mechanism. The current medications, drugs, and therapies for diseases and symptoms related abnormal increased muscle tone or excessive muscle contraction are aimed just at the pre-muscle molecular motor mechanisms, whereas without directly intending to effect on the muscle molecular motor mechanism i.e. the contractile apparatus mechanism at all, which, however, is in common for all kinds of muscle tissues. The muscle molecular motor mechanism mainly involves recycling of actin-myosin filament cross-bridge formation and sliding movement. In the process, bio-energy provided by ATP hydrolysis is necessarily required. Therefore, abnormal increased muscle tone or excessive contraction of muscle tissues under diseased conditions may be modified by **inhibition** of the muscle molecular motor with the actin-myosin ATPase inhibitor, which blocks hydrolysis of ATP, then reduces release of bio-energy for the muscle contraction.

Our studies in vitro and in vivo have demonstrated that BDM, an ATPase inhibitor, thereby, its analogues, derivatives, and other chemicals possessing similar effect on ATPase may be used as bio-energy muscle relaxants (general muscle relaxants).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2002:12580 USPATFULL
TITLE:	Bio-energy muscle relaxants

INVENTOR(S): Wang, Chong Gang, Montreal, CANADA
Zhang, Yisheng, Montreal, CANADA
Wang, Pei, Montreal, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002006962	A1	20020117
APPLICATION INFO.:	US 2001-764417	A1	20010119 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-180795P	20000207 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Yisheng Zhang and Pei Wang:, ADM Biotech, 1630 Du College, Saint-Laurent (Montreal), QC, H4L 2M4
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
LINE COUNT: 3596
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 5 OF 12 USPATFULL on STN
TI Therapeutic multispecific compounds comprised of anti-Fcalpha receptor antibodies
AB Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor antibodies and methods of use are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2001:133879 USPATFULL
TITLE: Therapeutic multispecific compounds comprised of anti-Fcalpha receptor antibodies
INVENTOR(S): Deo, Yashwant M., Audubon, PA, United States
Graziano, Robert, Frenchtown, NJ, United States
Keler, Tibor, Ottsville, PA, United States
PATENT ASSIGNEE(S): Mederax, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001014328	A1	20010816
APPLICATION INFO.:	US 2001-772120	A1	20010126 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1997-890011, filed on 10 Jul 1997, GRANTED, Pat. No. US 6193966
Continuation-in-part of Ser. No. US 1996-678194, filed on 11 Jul 1996, GRANTED, Pat. No. US 5922845
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS: 68
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 28 Drawing Page(s)
LINE COUNT: 2753
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 6 OF 12 USPATFULL on STN
TI T cell epitopes of the major allergens from dermatophagoides (house dust mite)
AB The present invention provides isolated peptides of the major protein allergens of the genus Dermatophagoides. Peptides within the scope of the invention comprises at least one T cell epitope, or preferably at least two T cell epitopes of a protein allergen selected from the allergens Der p I, Der p II, Der f I, or Der f II. The invention also pertains to modified peptides having similar or enhanced therapeutic properties as the corresponding, naturally-occurring allergen or portion thereof, but having reduced side effects. The invention further provides

nucleic acid sequences coding for peptides of the invention. Methods of treatment or of diagnosis of sensitivity to house dust mites in an individual and therapeutic compositions comprising one or more peptides of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:121598 USPATFULL
TITLE:	T cell epitopes of the major allergens from dermatophagoides (house dust mite)
INVENTOR(S):	Garman, Richard D., Arlington, MA, United States Greenstein, Julia L., West Newton, MA, United States Kuo, Mei-chang, Winchester, MA, United States Rogers, Bruce L., Belmont, MA, United States Franzen, Henry M., Watertown, MA, United States Chen, Xian, North Chelmsford, MA, United States Evans, Sean, Acton, MA, United States Shaked, Ze'ev, Berkeley, CA, United States
PATENT ASSIGNEE(S):	ImmuLogic Pharmaceutical Corporation, Waltham, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6268491	B1	20010731
APPLICATION INFO.:	US 1995-484296		19950607 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-445307, filed on 19 May 1995 Continuation-in-part of Ser. No. US 1994-227772, filed on 14 Apr 1994, now abandoned Continuation-in-part of Ser. No. WO 1993-US3471, filed on 14 Apr 1993 Continuation-in-part of Ser. No. US 1992-881396, filed on 8 May 1992, now abandoned Continuation-in-part of Ser. No. US 1991-777859, filed on 16 Oct 1991, now abandoned		

DOCUMENT TYPE:	Utility
FILE SEGMENT:	GRANTED
PRIMARY EXAMINER:	Scheiner, Laurie
LEGAL REPRESENTATIVE:	Lahive & Cockfield, LLP, Remillard, Esq., Jane E., Mandragouras, Esq., Amy E.
NUMBER OF CLAIMS:	5
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	61 Drawing Figure(s); 58 Drawing Page(s)
LINE COUNT:	4341

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 7 OF 12 USPATFULL on STN

TI Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor antibodies

AB Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor antibodies and methods of use are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:29120 USPATFULL
TITLE:	Therapeutic multispecific compounds comprised of anti-Fc.alpha. receptor antibodies
INVENTOR(S):	Deo, Yashwant M., Audubon, PA, United States Graziano, Robert, Frenchtown, NJ, United States Keler, Tibor, Ottsville, PA, United States
PATENT ASSIGNEE(S):	Mederax, Inc., Annandale, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6193966	B1	20010227
APPLICATION INFO.:	US 1997-890011		19970710 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-678194, filed		

on 11 Jul 1996, now patented, Pat. No. US 5922845
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Bansal, Geetha P.
LEGAL REPRESENTATIVE: Lahive & Cockfield, LLP, Remillard, Esq., Jane E.
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 30 Drawing Figure(s); 28 Drawing Page(s)
LINE COUNT: 2686
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 8 OF 12 USPATFULL on STN
TI Compositions and methods for regulation of active TNF-.alpha.
AB Substances comprising disaccharides and substances comprising carboxylated and/or sulfated oligosaccharides in substantially purified form, and methods of using same, are disclosed for the regulation of cytokine activity in a host. For instance, the secretion of active Tumor Necrosis Factor Alpha (TNF-.alpha.) can be either inhibited or augmented selectively by administration to the host of an effective amount of a substance of the invention. Thus, the present invention also relates to pharmaceutical compositions and their use for the prevention and/or treatment of pathological processes involving the induction of active cytokine secretion, such as TNF-.alpha.. The invention also relates to the initiation of a desirable immune system-related response by the host to the presence of activators, including pathogens. The substances and pharmaceutical compositions of the present invention may be administered daily, at very low effective doses, typically below 0.1 mg/kg human, or at intervals of up to about 5-8 days, preferably once a week.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2000:12786 USPATFULL
TITLE: Compositions and methods for regulation of active TNF-.alpha.
INVENTOR(S): Cohen, Irun R., Rehovot, Israel
Lider, Ofer, Rehovot, Israel
Cahalon, Liora, Givataim, Israel
Shoseyov, Oded, Shimshon, Israel
Margalit, Raanan, Rehovot, Israel
PATENT ASSIGNEE(S): Yeda Research and Development Co. Ltd., Rehovot, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6020323		20000201
APPLICATION INFO.:	US 1995-486127		19950607 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-436330, filed on 10 May 1995 which is a continuation-in-part of Ser. No. US 1993-96739, filed on 23 Jul 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-974750, filed on 10 Nov 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-878188, filed on 1 May 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Achutamurthy, Ponnathapura
ASSISTANT EXAMINER: Ponnaluri, P.
LEGAL REPRESENTATIVE: Pennie & Edmonds LLP
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 65 Drawing Figure(s); 54 Drawing Page(s)
LINE COUNT: 3440
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 9 OF 12 USPATFULL on STN

TI T cell epitopes of the major allergens from Dermatophagoides (house dust mite)

AB The present invention provides isolated peptides of the major protein allergens of the genus Dermatophagoides. Peptides within the scope of the invention comprises at least one T cell epitope, or preferably at least two T cell epitopes of a protein allergen selected from the allergens Der p I, Der p II, Der f I, or Der f II. The invention also pertains to modified peptides having similar or enhanced therapeutic properties as the corresponding, naturally-occurring allergen or portion thereof, but having reduced side effects. The invention further provides nucleic acid sequences coding for peptides of the invention. Methods of treatment or of diagnosis of sensitivity to house dust mites in an individual and therapeutic compositions comprising one or more peptides of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	1999:128144 USPATFULL
TITLE:	T cell epitopes of the major allergens from Dermatophagoides (house dust mite)
INVENTOR(S):	Garman, Richard D., Arlington, MA, United States Greenstein, Julia L., West Newton, MA, United States Kuo, Mei-chang, Winchester, MA, United States Rogers, Bruce L., Belmont, MA, United States Franzen, Henry M., Watertown, MA, United States Chen, Xian, North Chelmsford, MA, United States Evans, Sean, Acton, MA, United States Shaked, Ze'ev, Berkeley, CA, United States
PATENT ASSIGNEE(S):	Immulogic Pharamaceutical Corporation, Waltham, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5968526		19991019
APPLICATION INFO.:	US 1995-478572		19950607 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-445307, filed on 19 May 1995 which is a continuation-in-part of Ser. No. US 1994-227772, filed on 14 Apr 1994, now abandoned which is a continuation-in-part of Ser. No. WO 1995-US4481, filed on 12 Apr 1995		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Scheiner, Laurie		
LEGAL REPRESENTATIVE:	Lanive & Cockfield, LLP		
NUMBER OF CLAIMS:	13		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	58 Drawing Figure(s); 58 Drawing Page(s)		
LINE COUNT:	6649		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 10 OF 12 USPATFULL on STN

TI Methods for regulation of active TNF-.alpha.

AB Substances comprising disaccharides and substances comprising carboxylated and/or sulfated oligosaccharides in substantially purified form, and methods of using same, are disclosed for the regulation of cytokine activity in a host. For instance, the secretion of active Tumor Necrosis Factor Alpha (TNF-.alpha.) can be either inhibited or augmented selectively by administration to the host of an effective amount of a substance of the invention. Thus, the present invention also relates to pharmaceutical compositions and their use for the prevention and/or treatment of pathological processes involving the induction of active cytokine secretion, such as TNF-.alpha.. The invention also relates to the initiation of a desirable immune system-related response by the host to the presence of activators, including pathogens. The substances and pharmaceutical compositions of the present invention may be administered

daily, at very low effective doses, typically below 0.1 mg/kg human, or at intervals of tip to about 5-8 days, preferably once a week.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	1999:7369 USPATFULL		
TITLE:	Methods for regulation of active TNF-.alpha.		
INVENTOR(S):	Cohen, Irun R., Rehovot, Israel Lider, Ofer, Rehovot, Israel Cahalon, Liora, Givataim, Israel Shoseyov, Oded, Shimshon, Israel Margalit, Raanan, Rehovot, Israel		
PATENT ASSIGNEE(S):	Yeda Research and Development Co. Ltd., Israel (non-U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5861382		19990119
	WO 9411006		19940526
APPLICATION INFO.:	US 1995-436330		19950629 (8)
	WO 1993-US10868		19931109
			19950629 PCT 371 date
			19950629 PCT 102(e) date
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1993-96739, filed on 23 Jul 1993, now abandoned And a continuation-in-part of Ser. No. US 1992-974750, filed on 10 Nov 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-878188, filed on 1 May 1992, now abandoned And a continuation of Ser. No. US 1995-384203, filed on 3 Feb 1995, now patented, Pat. No. US 5474987		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Achutamurthy, Ponnathapura		
ASSISTANT EXAMINER:	Ponnaluri, Padmashri		
LEGAL REPRESENTATIVE:	Pennie & Edmonds LLP		
NUMBER OF CLAIMS:	5		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	65 Drawing Figure(s); 54 Drawing Page(s)		
LINE COUNT:	3391		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 11 OF 12 USPATFULL on STN

TI T cell epitopes of the major allergens from dermatophagoides (house dust mite)

AB The present invention provides isolated peptides of the major protein allergens of the genus Dermatophagoides. Peptides within the scope of the invention comprises at least one T cell epitope, or preferably at least two T cell epitopes of a protein allergen selected from the allergens Der p I, Der p II, Der f I, or Der f II. The invention also pertains to modified peptides having similar or enhanced therapeutic properties as the corresponding, naturally-occurring allergen or portion thereof, but having reduced side effects. The invention further provides nucleic acid sequences coding for peptides of the invention. Methods of treatment or of diagnosis of sensitivity to house dust mites in an individual and therapeutic compositions comprising one or more peptides of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	1998:124196 USPATFULL
TITLE:	T cell epitopes of the major allergens from dermatophagoides (house dust mite)
INVENTOR(S):	Garman, Richard D., Arlington, MA, United States Greenstein, Julia L., West Newton, MA, United States Kuo, Mei-chang, Winchester, MA, United States

Rogers, Bruce L., Belmont, MA, United States
Franzen, Henry M., Watertown, MA, United States
Chen, Xian, North Chelmsford, MA, United States
Evans, Sean, Acton, MA, United States
Shaked, Ze'ev, Berkeley, CA, United States
PATENT ASSIGNEE(S): Immulogic Pharmaceutical Corporation, Waltham, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5820862		19981013
APPLICATION INFO.:	US 1995-482142		19950607 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-445307, filed on 19 May 1995 which is a continuation-in-part of Ser. No. US 1994-227772, filed on 14 Apr 1994, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Scheiner, Laurie
LEGAL REPRESENTATIVE: Lahive & Cockfield, LLP
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 56 Drawing Figure(s); 58 Drawing Page(s)
LINE COUNT: 5621
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L18 ANSWER 12 OF 12 USPATFULL on STN
TI Method of production of antigen-specific glycosylation inhibiting factor
AB A method for the recombinant production and for the isolation of antigen-specific glycosylation inhibiting factor (AgGIF) is provided. Also disclosed is a method for modulating the immune responses in an antigen-specific manner utilizing a AgGIF, comprising soluble non-specific GIF-TCR.alpha. chains which bind to the antigen, and which suppress the immune response in an antigen-specific fashion.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 1998:111801 USPATFULL
TITLE: Method of production of antigen-specific glycosylation inhibiting factor
INVENTOR(S): Ishizaka, Kimishige, La Jolla, CA, United States
Ishii, Yasuyuki, La Jolla, CA, United States
PATENT ASSIGNEE(S): La Jolla Institute for Allergy and Immunology, San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5807714		19980915
APPLICATION INFO.:	US 1995-416336		19950404 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Eisenschenk, Frank C.
ASSISTANT EXAMINER: Nolan, Patrick
LEGAL REPRESENTATIVE: Fish & Richardson, P.C.
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 2069
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> s clostridium botulinum toxin
L19 443 CLOSTRIDIUM BOTULINUM TOXIN

=> s l19 and Fc fragment
L20 1 L19 AND FC FRAGMENT

=> d l20 ti abs ibib tot

L20 ANSWER 1 OF 1 USPATFULL on STN

TI Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof

AB A hybrid protein contains a protein that binds to a receptor of mastocytes and basophils and is endocyted by them. The protein can be IgE; IgE fragment; IgE **Fc fragment**; antibody against IgE receptor of mastocytes and basophils; fragment of the antibody against the IgE receptor of mastocytes and basophils; antibody against mastocyte specific potassium channel; and mast cell degranulating peptide. The hybrid protein also contains a protease cleaving proteins of the secretion process of the mastocytes and basophils so as to inhibit the secretion process without killing the mastocytes and basophils. The protease can be light chain **Clostridium botulinum toxin**; proteolytically active fragment of the light chain of a **Clostridium botulinum toxin** containing an amino acid sequence His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His wherein Xaa is an amino acid; light chain of the tetanus toxin; proteolytically active fragment of the light chain of the tetanus toxin containing His-Asp-Leu-Ile-His-Val-Leu-His; IgA protease of Neisseria gonorrhoeae; and proteolytic domain of the IgA protease of Neisseria gonorrhoeae.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:86306 USPATFULL		
TITLE:	Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof		
INVENTOR(S):	Bigalke, Hans, Hannover, GERMANY, FEDERAL REPUBLIC OF Frevert, Jurgen, Berlin, GERMANY, FEDERAL REPUBLIC OF		
PATENT ASSIGNEE(S):	BioteCon Gesellschaft fur biotechnologische Entwicklung und consulting mbH, Berlin, DE, 10589 (non-U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059912	A1	20030327
APPLICATION INFO.:	US 2002-64903	A1	20020827 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2001-700540, filed on 19 Jan 2001, PENDING A 371 of International Ser. No. WO 1999-EP3272, filed on 12 May 1999, UNKNOWN		
	NUMBER	DATE	
PRIORITY INFORMATION:	DE 1998-19821285	19980513	
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	GUDRUN E. HUCKETT, LONSSTR. 53, WUPPERTAL, 42289		
NUMBER OF CLAIMS:	11		
EXEMPLARY CLAIM:	1		
LINE COUNT:	576		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS' ENTERED AT 11:08:30 ON 01 DEC 2003

L1 1 S CONJUGATE AND MASTOCYTE BINDING
L2 171149 S HYBRID PROTEIN OR CONJUGATE
L3 21 S IGE AND IGA PROTEASE

```
L4          1711 S IGE AND TETANUS
L5             7 S L2 AND L3
L6             0 S L4 AND MASTOCYTE INACTIVATION
L7             0 S L4 AND DEGRANULATION INHIBITION
L8          1322 S MAST CELL DEGRANULATION AND INHIBITION
L9            38 S ALLERGY AND TREATEMENT
L10            0 S L9 AND L8
L11          107 S L8 AND ALLERGIC RESPONSE
L12           88 S L11 AND IGE
L13            2 S L12 AND TETANUS TOXIN
L14            1 S LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN
L15            0 S LIGHT CHAIN TETANUS TOXIN
L16            0 S IGA PROTEASE NEISSERIA GONORRHEAE
L17         1711 S TETANUS AND IGE
L18           12 S L8 AND L17
L19          443 S CLOSTRIDIUM BOTULINUM TOXIN
L20            1 S L19 AND FC FRAGMENT

=> s l19 and l8
L21            2 L19 AND L8

=> d l21 ti abs ibib tot

L21  ANSWER 1 OF 2  USPATFULL on STN
TI     Cytotoxin (non-neurotoxin) for the treatment of human headache disorders
       and inflammatory diseases
AB     Pharmaceutical applications of a chemodenervating agent reduce pain by
       altering release of pain- and inflammation-mediating autocoids, with a
       duration of action between 12-24 weeks. The limiting factor in dosing
       for this application is weakness and paralysis created by higher doses
       of the chemodenervating pharmaceutical mediated by action of the
       neurotoxin component of this chemodenervating pharmaceutical. The
       invention described herein represents a novel mechanism and
       pharmaceutical formulation which eliminates the neurotoxin component of
       the chemodenervating pharmaceutical, while retaining the cytotoxin
       component which provides an essential bioeffect for the relief of pain
       and inflammation. The invention allows for improvement in administering
       the pharmaceutical agent for the reduction of pain and/or inflammation
       without causing muscular weakness and paralysis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:       2002:329485  USPATFULL
TITLE:                  Cytotoxin (non-neurotoxin) for the treatment of human
                        headache disorders and inflammatory diseases
INVENTOR(S):            Borodic, Gary E., Canton, MA, UNITED STATES

                             NUMBER        KIND    DATE
                        ---------------- ------ --------
PATENT INFORMATION:     US 2002187164      A1   20021212
APPLICATION INFO.:      US 2002-212657     A1   20020805  (10)
RELATED APPLN. INFO.:   Continuation of Ser. No. US 1999-458784, filed on 10
                        Dec 1999, GRANTED, Pat. No. US 6429189
DOCUMENT TYPE:          Utility
FILE SEGMENT:           APPLICATION
LEGAL REPRESENTATIVE:   Michael N. Nitabach, Milbank, Tweed, Hadley & McCloy
                        LLP, 1 Chase Manhattan Plaza, New York, NY, 10005
NUMBER OF CLAIMS:       26
EXEMPLARY CLAIM:        1
NUMBER OF DRAWINGS:     8 Drawing Page(s)
LINE COUNT:             576
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21  ANSWER 2 OF 2  USPATFULL on STN
TI     Cytotoxin (non-neurotoxin) for the treatment of human headache disorders
```

and inflammatory diseases

AB Pharmaceutical applications of a chemodenervating agent reduce pain by altering release of pain and inflammation-mediating autocoids, with a duration of action between 12-24 weeks. The limiting factor in dosing for this application is weakness and paralysis created by higher doses of the chemodenervating pharmaceutical. This weakness and paralysis is mediated by action of the neurotoxin component of the chemodenervating pharmaceutical. The invention described herein represents a novel mechanism and pharmaceutical formulation which eliminates the neurotoxin component of the chemodenervating pharmaceutical, while retaining the cytotoxin component which provides an essential bioeffect for the relief of pain and inflammation. The invention allows for improvement in administering the pharmaceutical agent for the reduction of pain and/or inflammation without causing muscular weakness and paralysis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2002:194871 USPATFULL
TITLE:	Cytotoxin (non-neurotoxin) for the treatment of human headache disorders and inflammatory diseases
INVENTOR(S):	Borodic, Gary E., Canton, MA, United States
PATENT ASSIGNEE(S):	Botulinum Toxin Research Associates, Inc., Qunicy, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6429189	B1	20020806
APPLICATION INFO.:	US 1999-458784		19991210 (9)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	GRANTED
PRIMARY EXAMINER:	Cochrane Carlson, Karen
ASSISTANT EXAMINER:	Robinson, Hope A.
LEGAL REPRESENTATIVE:	Milbank, Tweed, Hadley & McCloy LLP
NUMBER OF CLAIMS:	29
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT:	758

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS' ENTERED AT 11:08:30 ON 01 DEC 2003

L1	1	S	CONJUGATE AND MASTOCYTE BINDING
L2	171149	S	HYBRID PROTEIN OR CONJUGATE
L3	21	S	IGE AND IGA PROTEASE
L4	1711	S	IGE AND TETANUS
L5	7	S	L2 AND L3
L6	0	S	L4 AND MASTOCYTE INACTIVATION
L7	0	S	L4 AND DEGRANULATION INHIBITION
L8	1322	S	MAST CELL DEGRANULATION AND INHIBITION
L9	38	S	ALLERGY AND TREATEMENT
L10	0	S	L9 AND L8
L11	107	S	L8 AND ALLERGIC RESPONSE
L12	88	S	L11 AND IGE
L13	2	S	L12 AND TETANUS TOXIN
L14	1	S	LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN
L15	0	S	LIGHT CHAIN TETANUS TOXIN
L16	0	S	IGA PROTEASE NEISSERIA GONORRHEAE
L17	1711	S	TETANUS AND IGE
L18	12	S	L8 AND L17
L19	443	S	CLOSTRIDIUM BOTULINUM TOXIN

```
L20             1 S L19 AND FC FRAGMENT
L21             2 S L19 AND L8

=> s mast cell degranulating peptide
L22           419 MAST CELL DEGRANULATING PEPTIDE

=> s l9 and l22
L23             0 L9 AND L22

=> d l22 ti abs ibib 1-10

L22  ANSWER 1 OF 419     MEDLINE on STN
TI   Cloning and characterization analysis of the genes encoding precursor of
     mast cell degranulating peptide from
     2 honeybee and 3 wasp species.
AB   The precursors of mast cell degranulating
     peptide (MCDP) genes were amplified by RT-PCR from the total RNA
     of venom gland of two honeybee species, Apis mellifera ligustica, Apis
     cerana cerana, and three wasp species, Vespa magnifica, Vespa velutina
     nigrothorax and Polistes hebraeus, respectively. Their PCR products were
     ligated into pGEM T-easy vector and the nucleotide sequences were
     analyzed. The length of five fragments was the same, it was 341 bp
     containing an ORF of 153 bp coding the precursor of MCDP and 188 bp 3'
     noncoding region. They have more than 90% homologues with each other in
     nucleotide sequences. The precursors of MCDP of A. cerana cerana, V.
     magnifica, V. velutina nigrothorax and P. hebraeus shared 96%, 100%, 94%
     and 98% homology with A. mellifera ligustica, respectively. The two
     species of wasps, V. magnifica and V. velutina nigrothorax, contained the
     same MCDP as A. mellifera ligustica, though they belong to different
     families with quite different biological properties, while A. cerana
     cerana contained the different MCDP in their venom as A. mellifera
     ligustica though they belong to the same genus. The fifth amino acid
     residue of MCDP in A. cerana cerana and P. hebraeus is arginine, replacing
     the cysteine, an important disulfide bridges element, in the position as
     in A. mellifera ligustica.
ACCESSION NUMBER:   2003500613     IN-PROCESS
DOCUMENT NUMBER:    22939087   PubMed ID: 14577379
TITLE:              Cloning and characterization analysis of the genes encoding
                    precursor of mast cell
                    degranulating peptide from 2 honeybee and
                    3 wasp species.
AUTHOR:             Zhang Su-Fang; Shi Wan-Jun; Cheng Jia-An; Zhang Chuan-Xi
CORPORATE SOURCE:   Institute of Applied Entomology, Zhejiang University,
                    Hangzhou 310029, China.. zhangsufang@fescomail.net
SOURCE:             I CHUAN HSUEH PAO. ACTA GENETICA SINICA, (2003 Sep) 30 (9)
                    861-6.
                    Journal code: 7900784. ISSN: 0379-4172.
PUB. COUNTRY:       China
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE:         Entered STN: 20031028
                    Last Updated on STN: 20031028

L22  ANSWER 2 OF 419     MEDLINE on STN
TI   Histamine-releasing activity and binding to the FcepsilonRI alpha human
     mast cell receptor subunit of mast cell
     degranulating peptide analogues with alanine
     substitutions.
AB   We have investigated the effects on mast cell binding and the
     histamine-releasing activity of l-alanine substitutions for the five
     lysine residues and the proline residue in the MCD peptide (1) sequence.
     All synthesized analogues Ala(2) (2), Ala(6) (3), Ala(11) (4), Ala(12)
     (5), Ala(17) (6), and Ala(21) (7) showed a loss of histamine release
```

compared to the parent MCD peptide 1. The order of decreased potency was 1 > 6 > 7 > 4 > 2 > 3 > 5. The alanine-substituted analogues showed a 5- to 6-fold decrease in histamine release for analogues 6, 7, and 4 and a 10-fold decrease for analogue 2. A more significant loss was observed in analogue 3 with a 75-fold loss of activity. The greatest loss of activity was observed with alanine substituting for proline in position 12. This analogue 5 showed a 130-fold loss of histamine release compared to the parent peptide 1. The ability of each analogue to interact with the FcepsilonRIalpha subunit of the human mast cell receptor was analyzed by competitive binding of the fluorescent peptide 1 and the alanine analogues using fluorescence polarization. The binding affinities of analogues 4, 6, and 7 for the mast cell receptor were less than the affinity of the native peptide 1. Analogues 2, 3, and 5 showed an increase in binding affinity, with analogue 5 showing the highest increase compared to the native peptide 1. The order of increased affinity was 5 > 3 > 2 > 1 > 4, 6, 7. On the basis of these results, the possibility that analogue 5 inhibits peptide 1-stimulated histamine release was examined. We found that peptide 5 did not inhibit histamine release by peptide 1. The analogues 2, 3, and especially analogue 5 may be useful leads toward study of agents that prevent binding of IgE to mast cell receptors.

ACCESSION NUMBER: 2003316290 MEDLINE
DOCUMENT NUMBER: 22710997 PubMed ID: 12825939
TITLE: Histamine-releasing activity and binding to the FcepsilonRI alpha human mast cell receptor subunit of **mast cell degranulating peptide** analogues with alanine substitutions.
AUTHOR: Buku A; Mendlowitz M; Condie B A; Price J A
CORPORATE SOURCE: Department of Physiology and Biophysics, Mount Sinai School of Medicine, 1 Gustave L Levy Place, Box 1218, New York, New York 10029, USA.. Angeliki.Buku@mssm.edu
SOURCE: JOURNAL OF MEDICINAL CHEMISTRY, (2003 Jul 3) 46 (14) 3008-12.
Journal code: 9716531. ISSN: 0022-2623.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 20030709
Last Updated on STN: 20030808
Entered Medline: 20030807

bad date

L22 ANSWER 3 OF 419 MEDLINE on STN
TI Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): a **mast cell degranulating peptide** mastoparan and phospholipase A1.
AB BACKGROUND: Venom sac extract of yellow jackets Vespula vulgaris was toxic in mice when injected intraperitoneally but not toxic when injected subcutaneously. Necropsy showed the toxicity to be an inflammatory response. METHODS: Venom peptide and protein fractions were tested to identify the inflammatory components. The active components were tested to establish whether they might function as adjuvant for venom protein-specific antibody response. RESULTS: Venom toxicity required the synergistic action of two venom components, a **mast cell degranulating peptide** mastoparan and phospholipase A1. Both components stimulated prostaglandin E(2) release from murine peritoneal cells and macrophages. Mastoparan showed a weak activity to enhance IgE and IgG1 responses to a yellow jacket venom protein Ves v 5 in BALB/c mice. It was not possible to assess the adjuvant activity of phospholipase A1 because of its suppression of Ves v 5-specific response. Melittin, a **mast cell degranulating peptide** from bee venom, was inactive as an adjuvant for Ves v 5-specific response. CONCLUSION: Yellow jacket venom contains two inflammatory components, mastoparan and phospholipase A1. Our findings

suggest that mastoparan can function as a weak adjuvant for TH2 cell-associated antibody response.
Copyright 2003 S. Karger AG, Basel

ACCESSION NUMBER: 2003235902 MEDLINE
DOCUMENT NUMBER: 22643038 PubMed ID: 12759486
TITLE: Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): a **mast cell degranulating peptide** mastoparan and phospholipase A1.
AUTHOR: King Te Piao; Jim Sui Yee; Wittkowski Knut M
CORPORATE SOURCE: The Rockefeller University, New York, NY 10021, USA.. kingtp@mail.rockefeller.edu
CONTRACT NUMBER: M01-RR00102 (NCRR)
SOURCE: INTERNATIONAL ARCHIVES OF ALLERGY AND IMMUNOLOGY, (2003 May) 131 (1) 25-32.
Journal code: 9211652. ISSN: 1018-2438.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200306
ENTRY DATE: Entered STN: 20030522
Last Updated on STN: 20030624
Entered Medline: 20030623

L22 ANSWER 4 OF 419 MEDLINE on STN
TI **Mast cell degranulating peptide** binds to RBL-2H3 mast cell receptors and inhibits IgE binding.
AB Fluorescent and biotinylated analogs of mast cell degranulating (MCD) peptide were synthesized and the labels fluoresceinisothiocyanate and N-hydroxysuccinimidobiotin were conjugated at position 1 in the MCD peptide sequence. The analogs with these moieties retained histamine-releasing activity as high as that of the parent MCD peptide in rat peritoneal mast cell assays. These labeled analogs were used in rat basophilic leukemia cells (RBL-2H3) to demonstrate by confocal microscopy and flow cytometry the specific binding of MCD peptide to mast cell receptors. Consequently MCD peptide was found to compete with and inhibit the binding of fluorescent IgE on RBL cells as monitored by flow cytometry. Thus MCD peptide may prove to be useful in the study of IgE receptor-bearing cells.

ACCESSION NUMBER: 2002061037 MEDLINE
DOCUMENT NUMBER: 21646709 PubMed ID: 11786182
TITLE: **Mast cell degranulating peptide** binds to RBL-2H3 mast cell receptors and inhibits IgE binding.
AUTHOR: Buku A; Price J A; Mendlowitz M; Masur S
CORPORATE SOURCE: Department of Physiology and Biophysics, Mount Sinai School of Medicine, New York, NY 10029, USA.. buku@physbio.mssm.edu
CONTRACT NUMBER: EY 09414 (NEI)
SOURCE: PEPTIDES, (2001 Dec) 22 (12) 1993-8.
Journal code: 8008690. ISSN: 0196-9781.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020314
Entered Medline: 20020313

L22 ANSWER 5 OF 419 MEDLINE on STN
TI Further studies on the structural requirements for mast cell degranulating (MCD) peptide-mediated histamine release.

AB Mast cell degranulating (MCD) peptide was modified in its two disulfide bridges and in the two arginine residues in order to measure the ability of these analogs to induce histamine release from mast cells in vitro. Analogs prepared were [Ala(3,15)]MCD, [Ala(5,19)]MCD, [Orn(16)]MCD, and [Orn(7,16)]MCD. Their histamine-releasing activity was determined spectrofluorometrically with peritoneal mast cells. The monocyclic analogs in which the cysteine residues were replaced pairwise with alanine residues showed three-to ten-fold diminished histamine-releasing activity respectively, compared with the parent MCD peptide. Substantial increases in activity were observed where arginine residues were replaced by ornithines. The ornithine-mono substituted analog showed an almost six-fold increase and the ornithine-doubly substituted analog three-fold increase in histamine-releasing activity compared with the parent MCD peptide. The structural changes associated with these activities were followed by circular dichroism (CD) spectroscopy. Changes in the shape and ellipticity of the CD spectra reflected a role for the disulfide bonds and the two arginine residues in the overall conformation and biological activity of the molecule.

ACCESSION NUMBER: 2002061036 MEDLINE
DOCUMENT NUMBER: 21646708 PubMed ID: 11786181
TITLE: Further studies on the structural requirements for mast cell degranulating (MCD) peptide-mediated histamine release.
AUTHOR: Buku A; Price J A
CORPORATE SOURCE: Department of Physiology and Biophysics, Mount Sinai School of Medicine, New York, NY 10029, USA.. buku@physbio.mssm.edu
SOURCE: PEPTIDES, (2001 Dec) 22 (12) 1987-91. Journal code: 8008690. ISSN: 0196-9781.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020314
Entered Medline: 20020313

L22 ANSWER 6 OF 419 MEDLINE on STN
TI A voltage-dependent transient K(+) current in rat dental pulp cells.
AB We characterized a voltage-dependent transient K(+) current in dental pulp fibroblasts on dental pulp slice preparations by using a nystatin perforated-patch recording configuration. The mean resting membrane potential of dental pulp fibroblasts was -53 mV. Depolarizing voltage steps to +60 mV from a holding potential of -80 mV evoked transient outward currents that are activated rapidly and subsequently inactivated during pulses. The activation threshold of the transient outward current was -40 mV. The reversal potential of the current closely followed the K(+) equilibrium potential, indicating that the current was selective for K(+). The steady-state inactivation of the peak outward K(+) currents described by a Boltzmann function with half-inactivation occurred at -47 mV. The K(+) current exhibited rapid activation, and the time to peak amplitude of the current was dependent on the membrane potentials. The inactivation process of the current was well fitted with a single exponential function, and the current exhibited slow inactivating kinetics (the time constants of decay ranged from 353 ms at -20 mV to 217 ms at +60 mV). The K(+) current was sensitive to intracellular Cs(+) and to extracellular 4-aminopyridine in a concentration-dependent manner, but it was not sensitive to tetraethylammonium, **mast cell degranulating peptide**, and dendrotoxin-I. The blood depressing substance-I failed to block the K(+) current. These results indicated that dental pulp fibroblasts expressed a slow-inactivating transient K(+) current.
ACCESSION NUMBER: 2001446713 MEDLINE

DOCUMENT NUMBER: 21385539 PubMed ID: 11492959
TITLE: A voltage-dependent transient K(+) current in rat dental pulp cells.
AUTHOR: Shibukawa Y; Suzuki T
CORPORATE SOURCE: Department of Physiology, Tokyo Dental College, Chiba, 261-8502 Japan.. yshibuka@tdc.ac.jp
SOURCE: JAPANESE JOURNAL OF PHYSIOLOGY, (2001 Jun) 51 (3) 345-53. Journal code: 2985184R. ISSN: 0021-521X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20010813
Last Updated on STN: 20011029
Entered Medline: 20011025

L22 ANSWER 7 OF 419 MEDLINE on STN
TI Crystallization and preliminary X-ray diffraction analysis of a eumenine mastoparan toxin: a new class of **mast-cell degranulating peptide** in the wasp venom.
AB Mastoparans are tetradecapeptides found to be the major component of vespid venoms. A mastoparan toxin isolated from the venom of Anterhynchium flavomarginatum micado has been crystallized and X-ray diffraction data collected to 2.7 A resolution using a synchrotron-radiation source. Crystals were determined to belong to the space group P6(2)22 (P6(4)22). This is the first mastoparan to be crystallized and will provide further insights into the conformational significance of mastoparan toxins with respect to their potency and activity in G-protein regulation.
ACCESSION NUMBER: 2001113651 MEDLINE
DOCUMENT NUMBER: 20508225 PubMed ID: 11053843
TITLE: Crystallization and preliminary X-ray diffraction analysis of a eumenine mastoparan toxin: a new class of **mast-cell degranulating peptide** in the wasp venom.
AUTHOR: Canduri F; Delatorre P; Fadel V; Lorenzi C C; Pereira J H; Olivieri J R; Ruggiero Neto J; Konno K; Palma M S; Yamane T; de Azevedo W F Jr
CORPORATE SOURCE: Departamento de Fisica, IBILCE, UNESP, CP 136, CEP 15054-000, Sao Jose Rio Preto, SP, Brazil.
SOURCE: ACTA CRYSTALLOGRAPHICA. SECTION D: BIOLOGICAL CRYSTALLOGRAPHY, (2000 Nov) 56 (Pt 11) 1434-6. Journal code: 9305878. ISSN: 0907-4449.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010215

L22 ANSWER 8 OF 419 MEDLINE on STN
TI Control of cell proliferation by cell volume alterations in rat C6 glioma cells.
AB K+ and Cl- channels are involved in regulating the proliferation of a number of cell types. Two main hypotheses have been proposed to explain the mechanism by which these channels influence cell proliferation: regulation of membrane potential and regulation of cell volume. In order to test these hypotheses, we measured, under different experimental conditions, the volume, membrane potential and rate of proliferation of C6 glioma cells. Cells cultured in control medium for 1-4 days were compared with cells cultured for the same period of time in the presence of broad

spectrum channel blockers: tetraethylammonium, 5-nitro-2-(3-phenylpropylamino) benzoic acid (NPPB) and Cs+, in hypertonic media (29% increased osmolarity with NaCl, KCl or sucrose), in hypotonic medium (23% decreased osmolarity with H2O) or in the presence of the specific channel blockers, i.e. **mast cell degranulating peptide**, charybdotoxin or chlorotoxin. In all of these conditions, we observed a close correspondence between the rate of proliferation and the mean cell volume. The proliferation decreased when volume increased. Moreover, whereas control cells were flattened, spindle-shaped, bipolar or multipolar, cells cultured in media supplemented with NPPB, KCl or CsCl were round with few processes. Of the agents tested, only KCl and Cs+ depolarized the cells. These results show that alterations of the rate of proliferation by K+ and Cl- channel blockers or anisotonia are closely related with changes in cell volume or form but are not correlated with changes in membrane potential.

ACCESSION NUMBER: 2000490281 MEDLINE
DOCUMENT NUMBER: 20494952 PubMed ID: 11041554
TITLE: Control of cell proliferation by cell volume alterations in rat C6 glioma cells.
AUTHOR: Rouzaire-Dubois B; Milandri J B; Bostel S; Dubois J M
CORPORATE SOURCE: Laboratoire de Neurobiologie Cellulaire et Moleculaire, CNRS UPR 9040, Gif-sur-Yvette, France.
SOURCE: PFLUGERS ARCHIV. EUROPEAN JOURNAL OF PHYSIOLOGY, (2000 Oct) 440 (6) 881-8.
Journal code: 0154720. ISSN: 0031-6768.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010201

L22 ANSWER 9 OF 419 MEDLINE on STN
TI Role of potassium channels in catecholamine secretion in the rat adrenal gland.
AB We elucidated the functional contribution of K(+) channels to cholinergic control of catecholamine secretion in the perfused rat adrenal gland. The small-conductance Ca(2+)-activated K(+) (SK(Ca))-channel blocker apamin (10-100 nM) enhanced the transmural electrical stimulation (ES; 1-10 Hz)- and 1, 1-dimethyl-4-phenyl-piperazinium (DMPP; 5-40 microM)-induced increases in norepinephrine (NE) output, whereas it did not affect the epinephrine (Epi) responses. Apamin enhanced the catecholamine responses induced by acetylcholine (6-200 microM) and methacholine (10-300 microM). The putative large-conductance Ca(2+)-activated K(+) channel blocker charybdotoxin (10-100 nM) enhanced the catecholamine responses induced by ES, but not the responses induced by cholinergic agonists. Neither the K(A) channel blocker **mast cell degranulating peptide** (100-1000 nM) nor the K(V) channel blocker margatoxin (10-100 nM) affected the catecholamine responses. These results suggest that SK(Ca) channels play an inhibitory role in adrenal catecholamine secretion mediated by muscarinic receptors and also in the nicotinic receptor-mediated secretion of NE, but not of Epi. Charybdotoxin-sensitive Ca(2+)-activated K(+) channels may control the secretion at the presynaptic site.

ACCESSION NUMBER: 2000411378 MEDLINE
DOCUMENT NUMBER: 20398462 PubMed ID: 10938231
TITLE: Role of potassium channels in catecholamine secretion in the rat adrenal gland.
AUTHOR: Nagayama T; Fukushima Y; Yoshida M; Suzuki-Kusaba M; Hisa H; Kimura T; Satoh S
CORPORATE SOURCE: Laboratory of Pharmacology, Graduate School of Pharmaceutical Sciences, Tohoku University, Aobayama,

Sendai, Japan.
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY. REGULATORY, INTEGRATIVE AND COMPARATIVE PHYSIOLOGY, (2000 Aug) 279 (2) R448-54.
Journal code: 100901230. ISSN: 0363-6119.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000907
Last Updated on STN: 20000907
Entered Medline: 20000831

L22 ANSWER 10 OF 419 MEDLINE on STN
TI Structure and biological activities of eumenine mastoparan-AF (EMP-AF), a new **mast cell degranulating peptide** in the venom of the solitary wasp (Anterhynchium flavomarginatum micado).
AB A new **mast cell degranulating peptide**, eumenine mastoparan-AF (EMP-AF), was isolated from the venom of the solitary wasp Anterhynchium flavomarginatum micado, the most common eumenine wasp found in Japan. The structure was analyzed by FAB-MS/MS together with Edman degradation, which was corroborated by solid-phase synthesis. The sequence of EMP-AF, Ile-Asn-Leu-Leu-Lys-Ile-Ala-Lys-Gly-Ile-Ile-Lys-Ser-Leu-NH(2), was similar to that of mastoparan, a **mast cell degranulating peptide** from a hornet venom; tetradecapeptide with C-terminus amidated and rich in hydrophobic and basic amino acids. In fact, EMP-AF exhibited similar activity to mastoparan in stimulating degranulation from rat peritoneal mast cells and RBL-2H3 cells. It also showed significant hemolytic activity in human erythrocytes. Therefore, this is the first example that a **mast cell degranulating peptide** is found in the solitary wasp venom. Besides the degranulation and hemolytic activity, EMP-AF also affects on neuromuscular transmission in the lobster walking leg preparation. Three analogs EMP-AF-1 approximately 3 were snythesized and biologically tested together with EMP-AF, resulting in the importance of the C-terminal amide structure for biological activities.
ACCESSION NUMBER: 2000407658 MEDLINE
DOCUMENT NUMBER: 20240153 PubMed ID: 10775751
TITLE: Structure and biological activities of eumenine mastoparan-AF (EMP-AF), a new **mast cell degranulating peptide** in the venom of the solitary wasp (Anterhynchium flavomarginatum micado).
AUTHOR: Konno K; Hisada M; Naoki H; Itagaki Y; Kawai N; Miwa A; Yasuhara T; Morimoto Y; Nakata Y
CORPORATE SOURCE: Institute of Biosciences of Rio Claro, Sao Paulo State University (UNESP), Rio Claro, Brazil.. kk-gon@rc.unesp.br
SOURCE: TOXICON, (2000 Nov) 38 (11) 1505-15.
Journal code: 1307333. ISSN: 0041-0101.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000901
Last Updated on STN: 20000901
Entered Medline: 20000822

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS'

```
     ENTERED AT 11:08:30 ON 01 DEC 2003
L1             1 S CONJUGATE AND MASTOCYTE BINDING
L2        171149 S HYBRID PROTEIN OR CONJUGATE
L3            21 S IGE AND IGA PROTEASE
L4          1711 S IGE AND TETANUS
L5             7 S L2 AND L3
L6             0 S L4 AND MASTOCYTE INACTIVATION
L7             0 S L4 AND DEGRANULATION INHIBITION
L8          1322 S MAST CELL DEGRANULATION AND INHIBITION
L9            38 S ALLERGY AND TREATEMENT
L10            0 S L9 AND L8
L11          107 S L8 AND ALLERGIC RESPONSE
L12           88 S L11 AND IGE
L13            2 S L12 AND TETANUS TOXIN
L14            1 S LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN
L15            0 S LIGHT CHAIN TETANUS TOXIN
L16            0 S IGA PROTEASE NEISSERIA GONORRHEAE
L17         1711 S TETANUS AND IGE
L18           12 S L8 AND L17
L19          443 S CLOSTRIDIUM BOTULINUM TOXIN
L20            1 S L19 AND FC FRAGMENT
L21            2 S L19 AND L8
L22          419 S MAST CELL DEGRANULATING PEPTIDE
L23            0 S L9 AND L22

=> s l22 and IgE
L24           19 L22 AND IGE

=> d 124 ti abs ibib tot

L24  ANSWER 1 OF 19      MEDLINE on STN
TI   Histamine-releasing activity and binding to the FcepsilonRI alpha human
     mast cell receptor subunit of mast cell
     degranulating peptide analogues with alanine
     substitutions.
AB   We have investigated the effects on mast cell binding and the
     histamine-releasing activity of l-alanine substitutions for the five
     lysine residues and the proline residue in the MCD peptide (1) sequence.
     All synthesized analogues Ala(2) (2), Ala(6) (3), Ala(11) (4), Ala(12)
     (5), Ala(17) (6), and Ala(21) (7) showed a loss of histamine release
     compared to the parent MCD peptide 1.  The order of decreased potency was
     1 > 6 > 7 > 4 > 2 > 3 > 5.  The alanine-substituted analogues showed a 5-
     to 6-fold decrease in histamine release for analogues 6, 7, and 4 and a
     10-fold decrease for analogue 2.  A more significant loss was observed in
     analogue 3 with a 75-fold loss of activity.  The greatest loss of activity
     was observed with alanine substituting for proline in position 12.  This
     analogue 5 showed a 130-fold loss of histamine release compared to the
     parent peptide 1.  The ability of each analogue to interact with the
     FcepsilonRIalpha subunit of the human mast cell receptor was analyzed by
     competitive binding of the fluorescent peptide 1 and the alanine analogues
     using fluorescence polarization.  The binding affinities of analogues 4,
     6, and 7 for the mast cell receptor were less than the affinity of the
     native peptide 1.  Analogues 2, 3, and 5 showed an increase in binding
     affinity, with analogue 5 showing the highest increase compared to the
     native peptide 1.  The order of increased affinity was 5 > 3 > 2 > 1 > 4,
     6, 7.  On the basis of these results, the possibility that analogue 5
     inhibits peptide 1-stimulated histamine release was examined.  We found
     that peptide 5 did not inhibit histamine release by peptide 1.  The
     analogues 2, 3, and especially analogue 5 may be useful leads toward study
     of agents that prevent binding of IgE to mast cell receptors.
ACCESSION NUMBER:    2003316290      MEDLINE
DOCUMENT NUMBER:     22710997   PubMed ID: 12825939
TITLE:               Histamine-releasing activity and binding to the FcepsilonRI
                     alpha human mast cell receptor subunit of mast
```

cell degranulating peptide
analogues with alanine substitutions.

AUTHOR: Buku A; Mendlowitz M; Condie B A; Price J A
CORPORATE SOURCE: Department of Physiology and Biophysics, Mount Sinai School of Medicine, 1 Gustave L Levy Place, Box 1218, New York, New York 10029, USA.. Angeliki.Buku@mssm.edu
SOURCE: JOURNAL OF MEDICINAL CHEMISTRY, (2003 Jul 3) 46 (14) 3008-12.
Journal code: 9716531. ISSN: 0022-2623.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 20030709
Last Updated on STN: 20030808
Entered Medline: 20030807

L24 ANSWER 2 OF 19 MEDLINE on STN
TI Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): a **mast cell degranulating peptide** mastoparan and phospholipase A1.
AB BACKGROUND: Venom sac extract of yellow jackets Vespula vulgaris was toxic in mice when injected intraperitoneally but not toxic when injected subcutaneously. Necropsy showed the toxicity to be an inflammatory response. METHODS: Venom peptide and protein fractions were tested to identify the inflammatory components. The active components were tested to establish whether they might function as adjuvant for venom protein-specific antibody response. RESULTS: Venom toxicity required the synergistic action of two venom components, a **mast cell degranulating peptide** mastoparan and phospholipase A1. Both components stimulated prostaglandin E(2) release from murine peritoneal cells and macrophages. Mastoparan showed a weak activity to enhance **IgE** and IgG1 responses to a yellow jacket venom protein Ves v 5 in BALB/c mice. It was not possible to assess the adjuvant activity of phospholipase A1 because of its suppression of Ves v 5-specific response. Melittin, a **mast cell degranulating peptide** from bee venom, was inactive as an adjuvant for Ves v 5-specific response. CONCLUSION: Yellow jacket venom contains two inflammatory components, mastoparan and phospholipase A1. Our findings suggest that mastoparan can function as a weak adjuvant for TH2 cell-associated antibody response.

ACCESSION NUMBER: 2003235902 MEDLINE
DOCUMENT NUMBER: 22643038 PubMed ID: 12759486
TITLE: Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): a **mast cell degranulating peptide** mastoparan and phospholipase A1.
AUTHOR: King Te Piao; Jim Sui Yee; Wittkowski Knut M
CORPORATE SOURCE: The Rockefeller University, New York, NY 10021, USA.. kingtp@mail.rockefeller.edu
CONTRACT NUMBER: M01-RR00102 (NCRR)
SOURCE: INTERNATIONAL ARCHIVES OF ALLERGY AND IMMUNOLOGY, (2003 May) 131 (1) 25-32.
Journal code: 9211652. ISSN: 1018-2438.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200306
ENTRY DATE: Entered STN: 20030522
Last Updated on STN: 20030624
Entered Medline: 20030623

L24 ANSWER 3 OF 19 MEDLINE on STN
TI **Mast cell degranulating peptide**
binds to RBL-2H3 mast cell receptors and inhibits **IgE** binding.
AB Fluorescent and biotinylated analogs of mast cell degranulating (MCD) peptide were synthesized and the labels fluoresceinisothiocyanate and N-hydroxysuccinimidobiotin were conjugated at position 1 in the MCD peptide sequence. The analogs with these moieties retained histamine-releasing activity as high as that of the parent MCD peptide in rat peritoneal mast cell assays. These labeled analogs were used in rat basophilic leukemia cells (RBL-2H3) to demonstrate by confocal microscopy and flow cytometry the specific binding of MCD peptide to mast cell receptors. Consequently MCD peptide was found to compete with and inhibit the binding of fluorescent **IgE** on RBL cells as monitored by flow cytometry. Thus MCD peptide may prove to be useful in the study of **IgE** receptor-bearing cells.

ACCESSION NUMBER: 2002061037 MEDLINE
DOCUMENT NUMBER: 21646709 PubMed ID: 11786182
TITLE: **Mast cell degranulating peptide** binds to RBL-2H3 mast cell receptors and inhibits **IgE** binding.
AUTHOR: Buku A; Price J A; Mendlowitz M; Masur S
CORPORATE SOURCE: Department of Physiology and Biophysics, Mount Sinai School of Medicine, New York, NY 10029, USA..
buku@physbio.mssm.edu
CONTRACT NUMBER: EY 09414 (NEI)
SOURCE: PEPTIDES, (2001 Dec) 22 (12) 1993-8.
Journal code: 8008690. ISSN: 0196-9781.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020314
Entered Medline: 20020313

L24 ANSWER 4 OF 19 MEDLINE on STN
TI Peptidergic pathway in human skin and rat peritoneal mast cell activation.
AB The common pathway of heterogenous mast cell activation as mediated by antigens is through the cross-linking of **IgE** bound to Fc epsilon RI receptors. The peptidergic pathway of mast cell activation, achieved by cationic secretagogues, is restricted to "serosal" mast cells, the experimental models being rat peritoneal and human skin mast cells. Cationic secretagogues include positively charged peptides but also various amines such as compound 48/80 and natural polyamines. An early intracellular event of this pathway is the activation of pertussis toxin-sensitive G proteins. The correlation observed between the ability of basic compounds to trigger mast cell exocytosis and their potency to activate purified G proteins strongly suggests that cationic compounds activate mast cell G proteins via a receptor-independent but membrane-assisted process. In this paper, alternative mechanisms are discussed. The consequence of G protein stimulation is the activation of phospholipase C with an increase in inositol triphosphates. Natural polyamines are relatively poor triggers of mast cells (10(-4) to 10(-2) M). Neuropeptides such as substance P, neuropeptide Y or vasoactive intestinal peptide, peptidic hormones such as kinins, and venoms such as mastoparan and **mast cell degranulating peptide**, are all active in a concentration range from 10(-7) to 10(-4) M. The cationic anaphylatoxin C3a also stimulates mast cells at concentrations below precursor complement C3 blood levels. The component C3 of the complement system is one of only a few plasma proteins having activation fragments (i.e. C3a) that can be generated at micromolar levels. The effects of basic secretagogues defines a peptidergic pathway

of mast cell activation, which represents a potentially toxic process considering the tissue effects caused by exogenous basic compounds such as venom peptides and certain amine containing drugs. Peptidergic activation of mast cells may also be a pathophysiological process having an important role in neurogenic inflammation and in diseases involving extensive activation of the blood complement cascade.

ACCESSION NUMBER: 94266602 MEDLINE
DOCUMENT NUMBER: 94266602 PubMed ID: 7515863
TITLE: Peptidergic pathway in human skin and rat peritoneal mast cell activation.
AUTHOR: Mousli M; Hugli T E; Landry Y; Bronner C
CORPORATE SOURCE: Laboratoire de Neuroimmunopharmacologie, INSERM CJF-9105, Universite Louis Pasteur-Strasbourg I, Illkirch, France.
SOURCE: IMMUNOPHARMACOLOGY, (1994 Jan-Feb) 27 (1) 1-11. Ref: 69
Journal code: 7902474. ISSN: 0162-3109.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940721
Last Updated on STN: 20000303
Entered Medline: 19940712

L24 ANSWER 5 OF 19 MEDLINE on STN
TI Purification of Ascaris suum antigen: its allergenic activity in vitro and in vivo.
AB Crude aqueous extracts of Ascaris suum (CE) have been used widely to study **IgE**-mediated reactions in various experimental preparations. Because some CE may contain a polypeptide, a **mast cell degranulating peptide** (MCDP), that degranulates mast cells by nonimmunologic mechanisms, various protocols have been used to ensure that the Ascaris preparation used did not contain MCPD. In general, these protocols have assumed MCDP had been without providing proof. Even protocols designed to isolate the major antigenic determinants from CE have usually been designed to evaluate immunogenic characteristics of the purified Ascaris; thus, few systematic comparisons of CE with purified Ascaris exist concerning mast cell degranulation, and few studies have demonstrated that MCDP has been removed during purification. Since Ascaris has proved to be useful in a variety of studies of **IgE**-mediated reactions, particularly in large animals (dog and sheep), we have developed a protocol to purify CE and MCDP and characterize their physiochemical and immunologic properties. We compared the allergenic activity of our purified Ascaris to that of CE and MCDP in skin and lung of natively sensitized dogs and in unsensitized rat peritoneal mast cells. Our results indicate that MCDP probably contaminates CE by less than 1.0%. However, the biologic activity of MCDP in dog lung appears insignificant and probably contributes little to CE-induced reactions in doses of CE commonly used (less than or equal to 100 mg injected). If a purified Ascaris preparation is essential, our protocol will yield an Ascaris preparation that has potent **IgE**-mediated effects in dog preparations with insignificant contamination by MCDP.

ACCESSION NUMBER: 86141326 MEDLINE
DOCUMENT NUMBER: 86141326 PubMed ID: 2419382
TITLE: Purification of Ascaris suum antigen: its allergenic activity in vitro and in vivo.
AUTHOR: Greenspon L W; White J; Shields R L; Fugner A; Gold W M
CONTRACT NUMBER: HL-00535 (NHLBI)
HL-07159 (NHLBI)
HL-24136 (NHLBI)
SOURCE: JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1986 Mar) 77

(3) 443-51.
Journal code: 1275002. ISSN: 0091-6749.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198604
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860415

L24 ANSWER 6 OF 19 MEDLINE on STN
TI A comparison of histamine secretion from peritoneal mast cells of the rat and hamster.
AB Functional mast cells have been obtained by peritoneal lavage of the rat and hamster. Both cell types released histamine on stimulation with appropriate dilutions of anti-rat **IgE** and anti-hamster serum. The maximum response evoked by each reagent was significantly greater for the hamster cells. The release was non-cytotoxic and was in each case blocked by the corresponding soluble antigen. The rat and hamster cells responded to concanavalin A and the lectin from lentil. Phosphatidylserine (PS) potentiated the release only from the rat cells. In the absence of the lipid, the hamster cells were more reactive. The lectin from wheat germ, in the presence of PS, evoked histamine secretion only from the rat cells. Both populations were refractory to the lectin from soybean and to protein A. Rat peritoneal cells were more responsive to the basic secretagogues compound 48/80 and peptide 401 (the MCD-peptide from bee venom). These differences were less marked in the case of polylysine and polyarginine. The two cell populations responded to the calcium ionophores A23187, ionomycin and chlortetracycline. The hamster cells were significantly more sensitive to the former two liberators but markedly less reactive to chlortetracycline. Adenosine 5'-triphosphate (ATP) and dextran were potent histamine liberators from the rat cells but were totally ineffective against the hamster. Acetylcholine and carbamylcholine had no effect on either cell type. These results are discussed in terms of the functional heterogeneity of mast cells from different sources.
ACCESSION NUMBER: 84204390 MEDLINE
DOCUMENT NUMBER: 84204390 PubMed ID: 6202354
TITLE: A comparison of histamine secretion from peritoneal mast cells of the rat and hamster.
AUTHOR: Leung K B; Pearce F L
SOURCE: BRITISH JOURNAL OF PHARMACOLOGY, (1984 Apr) 81 (4) 693-701.
Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198407
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19980206
Entered Medline: 19840718

L24 ANSWER 7 OF 19 MEDLINE on STN
TI [Bee venom allergy (a model of an **IgE**-mediated immediate-type allergy)].
Die Bienengiftallergie (Modell einer **IgE**-mediierten Soforttypallergie).
ACCESSION NUMBER: 81179063 MEDLINE
DOCUMENT NUMBER: 81179063 PubMed ID: 7013279
TITLE: [Bee venom allergy (a model of an **IgE**-mediated immediate-type allergy)].
Die Bienengiftallergie (Modell einer **IgE** -mediierten Soforttypallergie).

AUTHOR: Jarisch R
SOURCE: WIENER KLINISCHE WOCHENSCHRIFT. SUPPLEMENTUM, (1980) 122 3-27. Ref: 175
Journal code: 0357046. ISSN: 0300-5178.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198106
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19980206
Entered Medline: 19810623

L24 ANSWER 8 OF 19 USPATFULL on STN

TI Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof

AB A hybrid protein contains a protein that binds to a receptor of mastocytes and basophils and is endocyted by them. The protein can be **IgE**; **IgE** fragment; **IgE** Fc fragment; antibody against **IgE** receptor of mastocytes and basophils; fragment of the antibody against the **IgE** receptor of mastocytes and basophils; antibody against mastocyte specific potassium channel; and **mast cell degranulating peptide**.
The hybrid protein also contains a protease cleaving proteins of the secretion process of the mastocytes and basophils so as to inhibit the secretion process without killing the mastocytes and basophils. The protease can be light chain Clostridium botulinum toxin; proteolytically active fragment of the light chain of a Clostridium botulinum toxin containing an amino acid sequence His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His wherein Xaa is an amino acid; light chain of the tetanus toxin; proteolytically active fragment of the light chain of the tetanus toxin containing His-Asp-Leu-Ile-His-Val-Leu-His; IgA protease of Neisseria gonorrhoeae; and proteolytic domain of the IgA protease of Neisseria gonorrhoeae.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:86306 USPATFULL
TITLE: Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof
INVENTOR(S): Bigalke, Hans, Hannover, GERMANY, FEDERAL REPUBLIC OF
Frevert, Jurgen, Berlin, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): BioteCon Gesellschaft fur biotechnologische Entwicklung und consulting mbH, Berlin, DE, 10589 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059912	A1	20030327
APPLICATION INFO.:	US 2002-64903	A1	20020827 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-700540, filed on 19 Jan 2001, PENDING A 371 of International Ser. No. WO 1999-EP3272, filed on 12 May 1999, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	DE 1998-19821285	19980513

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GUDRUN E. HUCKETT, LONSSTR. 53, WUPPERTAL, 42289
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
LINE COUNT: 576
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L24 ANSWER 9 OF 19 USPATFULL on STN
TI Stabilized nanoparticle formulations of camptotheca derivatives
AB Pharmaceutical formulations are provided that increase the systemic bioavailability of camptotheca derivatives; preferably, the camptothecin derivative is 7-ethyl-10-hydroxyl camptothecin, SN-38. The drug is complexed with a stabilizing agent, but is not covalently bound thereto. Anionic or neutral lipids and/or polymers are used as the stabilizing agent, and secondary stabilizing agents and/or other excipients may be incorporated into the formulation as well. Therapeutic methods are also provided, wherein a formulation of the invention is administered to a patient to treat a condition, disorder, or disease that is responsive to camptothecin derivatives. Generally, administration is oral or parenteral.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:85861 USPATFULL
TITLE:	Stabilized nanoparticle formulations of camptotheca derivatives
INVENTOR(S):	Unger, Evan C., Tucson, AZ, UNITED STATES Romanowski, Marek J., Tucson, AZ, UNITED STATES Ramaswami, VaradaRajan, Tucson, AZ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059465	A1	20030327
APPLICATION INFO.:	US 2002-165867	A1	20020606 (10)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-703484, filed on 31 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2000-478124, filed on 5 Jan 2000, PENDING Continuation-in-part of Ser. No. US 1998-75477, filed on 11 May 1998, PENDING
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	REED & ASSOCIATES, 800 MENLO AVENUE, SUITE 210, MENLO PARK, CA, 94025
NUMBER OF CLAIMS:	55
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	2 Drawing Page(s)
LINE COUNT:	1903

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L24 ANSWER 10 OF 19 USPATFULL on STN
TI Preparation for the application of agents in mini-droplets
AB The invention relates to a preparation for the application of agents in the form of minuscule droplets of fluid, in particular provided with membrane-like structures consisting of one or several layers of amphiphilic molecules, or an amphiphilic carrier substance, in particular for transporting the agent into and through natural barriers such as skin and similar materials. The preparation contains a concentration of edge active substances which amounts to up to 99 mol-% of the agent concentration which is required for the induction of droplet solubilization. Such preparations are suitable, for example, for the non-invasive applications of antidiabetics, in particular of insulin. The invention, moreover, relates to the methods for the preparation of such formulations.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2000:174129 USPATFULL
TITLE:	Preparation for the application of agents in mini-droplets
INVENTOR(S):	Cevc, Gregor, Heimstetten, Germany, Federal Republic of
PATENT ASSIGNEE(S):	Idea AG, Munich, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6165500		20001226	
APPLICATION INFO.:	US 1992-844664		19920408	(7)

	NUMBER	DATE
PRIORITY INFORMATION:	DE 1990-4026834	19900824
	DE 1990-4026833	19900824
	DE 1991-4107153	19910306
	WO 1991-EP1596	19910822

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kishore, Gollamudi S.
LEGAL REPRESENTATIVE: Davidson, Davidson & Kappel, LLC
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 31 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 4336
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L24 ANSWER 11 OF 19 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Histamine-releasing activity and binding to the Fc.epsilon.RI.alpha. human mast cell receptor subunit of **mast cell degranulating peptide** analogues with alanine substitutions.

AB We have investigated the effects on mast cell binding and the histamine-releasing activity of L-alanine substitutions for the five lysine residues and the proline residue in the MCD peptide (1) sequence. All synthesized analogues Ala(2) (2), Ala(6) (3), Ala(11) (4), Ala(12) (5), Ala(17) (6), and Ala(21) (7) showed a loss of histamine release compared to the parent MCD peptide 1. The order of decreased potency was 1 > 6 > 7 > 4 > 2 > 3 > 5. The alanine-substituted analogues showed a 5- to 6-fold decrease in histamine release for analogues 6, 7, and 4 and a 10-fold decrease for analogue 2. A more significant loss was observed in analogue 3 with a 75-fold loss of activity. The greatest loss of activity was observed with alanine substituting for proline in position 12. This analogue 5 showed a 130-fold loss of histamine release compared to the parent peptide 1. The ability of each analogue to interact with the Fc.epsilon.RI.alpha. subunit of the human mast cell receptor was analyzed by competitive binding of the fluorescent peptide 1 and the alanine analogues using fluorescence polarization. The binding affinities of analogues 4, 6, and 7 for the mast cell receptor were less than the affinity of the native peptide 1. Analogues 2, 3, and 5 showed an increase in binding affinity, with analogue 5 showing the highest increase compared to the native peptide 1. The order of increased affinity was 5 > 3 > 2 > 1 > 4, 6, 7. On the basis of these results, the possibility that analogue 5 inhibits peptide 1-stimulated histamine release was examined. We found that peptide 5 did not inhibit histamine release by peptide 1. The analogues 2, 3, and especially analogue 5 may be useful leads toward study of agents that prevent binding of **IgE** to mast cell receptors.

ACCESSION NUMBER: 2003259476 EMBASE
TITLE: Histamine-releasing activity and binding to the Fc.epsilon.RI.alpha. human mast cell receptor subunit of **mast cell degranulating peptide** analogues with alanine substitutions.
AUTHOR: Buku A.; Mendlowitz M.; Condie B.A.; Price J.A.
CORPORATE SOURCE: A. Buku, Dept. of Physiology and Biophysics, Mount Sinai School of Medicine, Box 1218, 1 Gustave L. Levy Place, New York, NY 10029, United States
SOURCE: Journal of Medicinal Chemistry, (3 Jul 2003) 46/14 (3008-3012).

Refs: 37
ISSN: 0022-2623 CODEN: JMCMAR
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

L24 ANSWER 12 OF 19 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): A **mast cell degranulating peptide** mastoparan and phospholipase A1.
AB Background: Venom sac extract of yellow jackets Vespula vulgariswas toxic in mice when injected intraperitoneally but not toxic when injected subcutaneously. Necropsy showed the toxicity to be an inflammatory response. Methods: Venom peptide and protein fractions were tested to identify the inflammatory components. The active components were tested to establish whether they might function as adjuvant for venom protein-specific antibody response. Results: Venom toxicity required the synergistic action of two venom components, a **mast cell degranulating peptide** mastoparan and phospholipase A1. Both components stimulated prostaglandin E(2) release from murine peritoneal cells and macrophages. Mastoparan showed a weak activity to enhance **IgE** and IgG1 responses to a yellow jacket venom protein Ves v 5 in BALB/c mice. It was not possible to assess the adjuvant activity of phospholipase A1 because of its suppression of Ves v 5-specific response. Melittin, a **mast cell degranulating peptide** from bee venom, was inactive as an adjuvant for Ves v 5-specific response. Conclusion: Yellow jacket venom contains two inflammatory components, mastoparan and phospholipase A1. Our findings suggest that mastoparan can function as a weak adjuvant for TH2 cell-associated antibody response. Copyright .COPYRGT. 2003 S. Karger AG, Basel.
ACCESSION NUMBER: 2003223194 EMBASE
TITLE: Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): A **mast cell degranulating peptide** mastoparan and phospholipase A1.
AUTHOR: King T.P.; Jim S.Y.; Wittkowski K.M.
CORPORATE SOURCE: Dr. T.P. King, Rockefeller University, 1230 York Avenue, New York, NY 10021, United States. kingtp@mail.rockefeller.edu
SOURCE: International Archives of Allergy and Immunology, (2003) 131/1 (25-32).
Refs: 36
ISSN: 1018-2438 CODEN: IAAIEG
COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English

L24 ANSWER 13 OF 19 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Peptidergic pathway in human skin and rat peritoneal mast cell activation.
AB The common pathway of heterogenous mast cell activation as mediated by antigens is through the cross-linking of **IgE** bound to Fc.epsilon.RI receptors. The peptidergic pathway of mast cell activation, achieved by cationic secretagogues, is restricted to 'serosal' mast cells, the experimental models being rat peritoneal and human skin mast cells.

Cationic secretagogues include positively charged peptides but also various amines such as compound 48/80 and natural polyamines. An early intracellular event of this pathway is the activation of pertussis toxin-sensitive G proteins. The correlation observed between the ability of basic compounds to trigger mast cell exocytosis and their potency to activate purified G proteins strongly suggests that cationic compounds activate mast cell G proteins via a receptor-independent but membrane-assisted process. In this paper, alternative mechanisms are discussed. The consequence of G protein stimulation is the activation of phospholipase C with an increase in inositol triphosphates. Natural polyamines are relatively poor triggers of mast cells (10-4 to 10-2 M). Neuropeptides such as substance P, neuropeptide Y or vasoactive intestinal peptide, peptidic hormones such as kinins, and venoms such as mastoparan and **mast cell degranulating peptide**
, are all active in a concentration range from 10-7 to 10-4 M. The cationic anaphylatoxin C3a also stimulates mast cells at concentrations below precursor complement C3 blood levels. The component C3 of the complement system is one of only a few plasma proteins having activation fragments (i.e. C3a) that can be generated at micromolar levels. The effects of basic secretagogues defines a peptidergic pathway of mast cell activation, which represents a potentially toxic process considering the tissue effects caused by exogenous basic compounds such as venom peptides and certain amine containing drugs. Peptidergic activation of mast cells may also be a pathophysiological process having an important role in neurogenic inflammation and in diseases involving extensive activation of the blood complement cascade.

ACCESSION NUMBER: 94045163 EMBASE
DOCUMENT NUMBER: 1994045163
TITLE: Peptidergic pathway in human skin and rat peritoneal mast cell activation.
AUTHOR: Mousli M.; Hugli T.E.; Landry Y.; Bronner C.
CORPORATE SOURCE: Lab. de Neuroimmunopharmacologie, INSERM CJF-9105, Univ. Louis Pasteur-Strasbourg I, B.P. 24,67401 Illkirch Cedex, France
SOURCE: Immunopharmacology, (1994) 27/1 (1-11).
ISSN: 0162-3109 CODEN: IMMUDP
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
013 Dermatology and Venereology
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English

TI Purification of Ascaris suum antigen: Its allergenic activity in vitro and in vivo.

AB Crude aqueous extracts of Ascaris suum (CE) have been used widely to study **IgE**-mediated reactions in various experimental preparations. Because some CE may contain a polypeptide, a **mast cell degranulating peptide** (MCDP), that degranulates mast cells by nonimmunologic mechanisms, various protocols have been used to ensure that the Ascaris preparation used did not contain MCPD. In general, these protocols have assumed MCDP had been removed without providing proof. Even protocols designed to isolate the major antigenic determinants from CE have usually been designed to evaluate immunogenic characteristics of the purified Ascaris; thus, few systematic comparisons of CE with purified Ascaris exist concerning mast cell degranulation, and few studies have demonstrated that MCDP has been remove during purification. Since Ascaris has proved to be useful in a variety of studies of **IgE** -mediated reactions, particularly in large animals (dog and sheep), we have developed a protocol to purify CE and MCDP and characterize their physiochemical and immunologic properties. We compared the allergenic

activity of our purified Ascaris to that of CE and MCDP in skin and lung of natively sensitized dogs and in unsensitized rat peritoneal mast cells. Our results indicate that MCDP probably contaminates CE by <1.0%. However, the biologic activity of MCDP in dog lung appears insignificant and probably contributes little to CE-induced reactions in doses of CE commonly used (.ltoreq.100 mg injected). If a purified Ascaris preparation is essential, our protocol will yield an Ascaris preparation that has potent **IgE**-mediated effects in dog preparations with insignificant contamination by MCDP.

ACCESSION NUMBER: 86124757 EMBASE
DOCUMENT NUMBER: 1986124757
TITLE: Purification of Ascaris suum antigen: Its allergenic activity in vitro and in vivo.
AUTHOR: Greenspon L.W.; White J.; Shields R.L.; et al.
CORPORATE SOURCE: Cardiovascular Research Institute, University of California, San Francisco, CA 94143, United States
SOURCE: Journal of Allergy and Clinical Immunology, (1986) 77/3 (443-451).
CODEN: JACIBY
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 013 Dermatology and Venereology
LANGUAGE: English

L24 ANSWER 15 OF 19 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
TI Histamine-releasing activity and binding to the FcepsilonRIalpha human mast cell receptor subunit of **mast cell degranulating peptide** analogues with alanine substitutions.
AB We have investigated the effects on mast cell binding and the histamine-releasing activity of L-alanine substitutions for the five lysine residues and the proline residue in the MCD peptide (1) sequence. All synthesized analogues Ala2 (2), Ala6 (3), Ala11 (4), Ala12 (5), Ala17 (6), and Ala21 (7) showed a loss of histamine release compared to the parent MCD peptide 1. The order of decreased potency was 1>6>7>4>2>3>5. The alanine-substituted analogues showed a 5- to 6-fold decrease in histamine release for analogues 6, 7, and 4 and a 10-fold decrease for analogue 2. A more significant loss was observed in analogue 3 with a 75-fold loss of activity. The greatest loss of activity was observed with alanine substituting for proline in position 12. This analogue 5 showed a 130-fold loss of histamine release compared to the parent peptide 1. The ability of each analogue to interact with the FcepsilonRIalpha subunit of the human mast cell receptor was analyzed by competitive binding of the fluorescent peptide 1 and the alanine analogues using fluorescence polarization. The binding affinities of analogues 4, 6, and 7 for the mast cell receptor were less than the affinity of the native peptide 1. Analogues 2, 3, and 5 showed an increase in binding affinity, with analogue 5 showing the highest increase compared to the native peptide 1. The order of increased affinity was 5>3>2>1>4, 6, 7. On the basis of these results, the possibility that analogue 5 inhibits peptide 1-stimulated histamine release was examined. We found that peptide 5 did not inhibit histamine release by peptide 1. The analogues 2, 3, and especially analogue 5 may be useful leads toward study of agents that prevent binding of **IgE** to mast cell receptors.

ACCESSION NUMBER: 2003:438594 BIOSIS
DOCUMENT NUMBER: PREV200300438594
TITLE: Histamine-releasing activity and binding to the FcepsilonRIalpha human mast cell receptor subunit of **mast cell degranulating peptide** analogues with alanine substitutions.
AUTHOR(S): Buku, A. [Reprint Author]; Mendlowitz, M.; Condie, B. A.; Price, J. A.
CORPORATE SOURCE: Department of Physiology and Biophysics, Mount Sinai School of Medicine, 1 Gustave L. Levy Place, Box 1218, New York,

NY, 10029, USA
Angeliki.Buku@mssm.edu
SOURCE: Journal of Medicinal Chemistry, (July 3 2003) Vol. 46, No. 14, pp. 3008-3012. print.
ISSN: 0022-2623 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Sep 2003
Last Updated on STN: 24 Sep 2003

L24 ANSWER 16 OF 19 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
TI Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): A **mast cell degranulating peptide** mastoparan and phospholipase A1.
AB Background: Venom sac extract of yellow jackets Vespula vulgaris was toxic in mice when injected intraperitoneally but not toxic when injected subcutaneously. Necropsy showed the toxicity to be an inflammatory response. Methods: Venom peptide and protein fractions were tested to identify the inflammatory components. The active components were tested to establish whether they might function as adjuvant for venom protein-specific antibody response. Results: Venom toxicity required the synergistic action of two venom components, a **mast cell degranulating peptide** mastoparan and phospholipase A1. Both components stimulated prostaglandin E2 release from murine peritoneal cells and macrophages. Mastoparan showed a weak activity to enhance **IgE** and IgG1 responses to a yellow jacket venom protein Ves v 5 in BALB/c mice. It was not possible to assess the adjuvant activity of phospholipase A1 because of its suppression of Ves v 5-specific response. Melittin, a **mast cell degranulating peptide** from bee venom, was inactive as an adjuvant for Ves v 5-specific response. Conclusion: Yellow jacket venom contains two inflammatory components, mastoparan and phospholipase A1. Our findings suggest that mastoparan can function as a weak adjuvant for TH2 cell-associated antibody response.
ACCESSION NUMBER: 2003:331578 BIOSIS
DOCUMENT NUMBER: PREV200300331578
TITLE: Inflammatory role of two venom components of yellow jackets (Vespula vulgaris): A **mast cell degranulating peptide** mastoparan and phospholipase A1.
AUTHOR(S): King, Te Piao [Reprint Author]; Jim, Sui Yee; Wittkowski, Knut M.
CORPORATE SOURCE: The Rockefeller University, 1230 York Avenue, New York, NY, 10021, USA
kingtp@mail.rockefeller.edu
SOURCE: International Archives of Allergy and Immunology, (May 2003) Vol. 131, No. 1, pp. 25-32. print.
CODEN: IAAIEG. ISSN: 1018-2438.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003

L24 ANSWER 17 OF 19 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
TI **Mast cell degranulating peptide** binds to RBL-2H3 mast cell receptors and inhibits **IgE** binding.
AB Fluorescent and biotinylated analogs of mast cell degranulating (MCD) peptide were synthesized and the labels fluoresceinisothiocyanate and N-hydroxysuccinimidobiotin were conjugated at position 1 in the MCD peptide sequence. The analogs with these moieties retained histamine-releasing activity as high as that of the parent MCD peptide in rat peritoneal mast cell assays. These labeled analogs were used in rat basophilic leukemia cells (RBL-2H3) to demonstrate by confocal microscopy and flow cytometry the specific binding of MCD peptide to mast cell

receptors. Consequently MCD peptide was found to compete with and inhibit the binding of fluorescent **IgE** on RBL cells as monitored by flow cytometry. Thus MCD peptide may prove to be useful in the study of **IgE** receptor-bearing cells.

ACCESSION NUMBER: 2002:160672 BIOSIS
DOCUMENT NUMBER: PREV200200160672
TITLE: **Mast cell degranulating peptide** binds to RBL-2H3 mast cell receptors and inhibits **IgE** binding.
AUTHOR(S): Buku, Angeliki [Reprint author]; Price, Joseph A.; Mendlowitz, Milton; Masur, Sandra
CORPORATE SOURCE: Department of Physiology and Biophysics, Mount Sinai School of Medicine, New York, NY, 10029, USA
buku@physbio.mssm.edu
SOURCE: Peptides (New York), (December, 2001) Vol. 22, No. 12, pp. 1993-1998. print.
CODEN: PPTDD5. ISSN: 0196-9781.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Feb 2002
Last Updated on STN: 26 Feb 2002

L24 ANSWER 18 OF 19 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
TI Peptidergic pathway in human skin and rat peritoneal mast cell activation.
AB The common pathway of heterogenous mast cell activation as mediated by antigens is through the cross-linking of **IgE** bound to Fc-epsilon-RI receptors. The peptidergic pathway of mast cell activation, achieved by cationic secretagogues, is restricted to "serosal" mast cells, the experimental models being rat peritoneal and human skin mast cells. Cationic secretagogues include positively charged peptides but also various amines such as compound 48/80 and natural polyamines. An early intracellular event of this pathway is the activation of pertussis toxin-sensitive G proteins. The correlation observed between the ability of basic compounds to trigger mast cell exocytosis and their potency to activate purified G proteins strongly suggests that cationic compounds activate mast cell G proteins via a receptor-independent but membrane-assisted process. In this paper, alternative mechanisms are discussed. The consequence of G protein stimulation is the activation of phospholipase C with an increase in inositol triphosphates. Natural polyamines are relatively poor triggers of mast cells (10-4 to 10-2 M). Neuropeptides such as substance P, neuropeptide Y or vasoactive intestinal peptide, peptidic hormones such as kinins, and venoms such as mastoparan and **mast cell degranulating peptide**, are all active in a concentration range from 10-7 to 10-4 M. The cationic anaphylatoxin C3a also stimulates mast cells at concentrations below precursor complement C3 blood levels. The component C3 of the complement system is one of only a few plasma proteins having activation fragments (i.e. C3a) that can be generated at micromolar levels. The effects of basic secretagogues defines a peptidergic pathway of mast cell activation, which represents a potentially toxic process considering the tissue effects caused by exogenous basic compounds such as venom peptides and certain amine containing drugs. Peptidergic activation of mast cells may also be a pathophysiological process having an important role in neurogenic inflammation and in diseases involving extensive activation of the blood complement cascade.

ACCESSION NUMBER: 1994:154344 BIOSIS
DOCUMENT NUMBER: PREV199497167344
TITLE: Peptidergic pathway in human skin and rat peritoneal mast cell activation.
AUTHOR(S): Mousli, M. [Reprint author]; Hugli, T. E.; Landry, Y.; Bronner, C.
CORPORATE SOURCE: Lab. de Neuroimmunopharmacologie, INSERM CJF-9105, Universite Louis Pasteur-Strasbourg I, B.P. 24, 67401 Illkirch Cedex, France

SOURCE: Immunopharmacology, (1994) Vol. 27, No. 1, pp. 1-11.
CODEN: IMMUDP. ISSN: 0162-3109.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Apr 1994
Last Updated on STN: 10 Apr 1994

L24 ANSWER 19 OF 19 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
TI PURIFICATION OF ASCARIS-SUUM ANTIGEN ITS ALLERGENIC ACTIVITY IN-VITRO AND IN-VIVO.
AB Crude aqueous extracts of Ascaris suum (CE) have been used widely to study **IgE**-mediated reactions in various experimental preparations. Because some CE may contain a polypeptide, a **mast cell degranulating peptide** (MCDP), that degranulates mast cells by nonimmunologic mechanisms, various protocols have been used to ensure that the Ascaris preparation used did not contain MCPD. In general, these protocols have assumed MCDP had been removed without providing proof. Even protocols designed to isolate the major antigenic determinants from CE have usually been designed to evaluate immunogenic characteristics of the purified Ascaris; thus, few systematic comparisons of CE with purified Ascaris exist concerning mass cell degranulation, and few studies have demonstrated that MCDP has been removed during purification. Since Ascaris has proved to be useful in a variety of studies of **IgE**-mediated reactions, particularly in large animals (dog and sheep), we have developed a protocol to purify CE and MCDP and characterize their physiochemical and immunologic properties. We compared the allergenic activity of our purified Ascaris to that of CE and MCDP in skin and lung of natively sensitized dogs and in unsensitized rat peritoneal mast cells. Our results indicate that MCDP probably contaminates CE by < 1.0%. However, the biological activity of MCDP in dog lung appears insignificant and probably contributes little to CE-induced reactions in doses of CE commonly used (.ltoreq. 100 mg injected). If a purified Ascaris preparation is essential, our protocol will yield an Ascaris preparation that has potent **IgE**-mediated effects in dog preparations with insignificant contamination by MCDP.
ACCESSION NUMBER: 1986:235748 BIOSIS
DOCUMENT NUMBER: PREV198682000252; BA82:252
TITLE: PURIFICATION OF ASCARIS-SUUM ANTIGEN ITS ALLERGENIC ACTIVITY IN-VITRO AND IN-VIVO.
AUTHOR(S): GREENSPON L W [Reprint author]; WHITE J; SHIELDS R L; FUEGNER A; GOLD W M
CORPORATE SOURCE: CARDIOVASCULAR RESEARCH INSTITUTE, 1327-M, UNIVERSITY OF CALIFORNIA, SAN FRANCISCO, SAN FRANCISCO, CALIF 94143, USA
SOURCE: Journal of Allergy and Clinical Immunology, (1986) Vol. 77, No. 3, pp. 443-451.
CODEN: JACIBY. ISSN: 0091-6749.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 7 Jun 1986
Last Updated on STN: 7 Jun 1986

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS' ENTERED AT 11:08:30 ON 01 DEC 2003
L1 1 S CONJUGATE AND MASTOCYTE BINDING
L2 171149 S HYBRID PROTEIN OR CONJUGATE
L3 21 S IGE AND IGA PROTEASE
L4 1711 S IGE AND TETANUS

```
L5             7 S L2 AND L3
L6             0 S L4 AND MASTOCYTE INACTIVATION
L7             0 S L4 AND DEGRANULATION INHIBITION
L8          1322 S MAST CELL DEGRANULATION AND INHIBITION
L9            38 S ALLERGY AND TREATEMENT
L10            0 S L9 AND L8
L11          107 S L8 AND ALLERGIC RESPONSE
L12           88 S L11 AND IGE
L13            2 S L12 AND TETANUS TOXIN
L14            1 S LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN
L15            0 S LIGHT CHAIN TETANUS TOXIN
L16            0 S IGA PROTEASE NEISSERIA GONORRHEAE
L17         1711 S TETANUS AND IGE
L18           12 S L8 AND L17
L19          443 S CLOSTRIDIUM BOTULINUM TOXIN
L20            1 S L19 AND FC FRAGMENT
L21            2 S L19 AND L8
L22          419 S MAST CELL DEGRANULATING PEPTIDE
L23            0 S L9 AND L22
L24           19 S L22 AND IGE

=> s l22 and l19
L25            1 L22 AND L19

=> d l25 ti abs ibib tot

L25  ANSWER 1 OF 1  USPATFULL on STN
TI     Hybrid protein for inhibiting the degranulation of mastocytes and the
       use thereof
AB     A hybrid protein contains a protein that binds to a receptor of
       mastocytes and basophils and is endocyted by them. The protein can be
       IgE; IgE fragment; IgE Fc fragment; antibody against IgE receptor of
       mastocytes and basophils; fragment of the antibody against the IgE
       receptor of mastocytes and basophils; antibody against mastocyte
       specific potassium channel; and mast cell
       degranulating peptide. The hybrid protein also
       contains a protease cleaving proteins of the secretion process of the
       mastocytes and basophils so as to inhibit the secretion process without
       killing the mastocytes and basophils. The protease can be light chain
       Clostridium botulinum toxin; proteolytically
       active fragment of the light chain of a Clostridium
       botulinum toxin containing an amino acid sequence
       His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His wherein Xaa is an amino acid; light
       chain of the tetanus toxin; proteolytically active fragment of the light
       chain of the tetanus toxin containing His-Asp-Leu-Ile-His-Val-Leu-His;
       IgA protease of Neisseria gonorrhoeae; and proteolytic domain of the IgA
       protease of Neisseria gonorrhoeae.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:        2003:86306  USPATFULL
TITLE:                   Hybrid protein for inhibiting the degranulation of
                         mastocytes and the use thereof
INVENTOR(S):             Bigalke, Hans, Hannover, GERMANY, FEDERAL REPUBLIC OF
                         Frevert, Jurgen, Berlin, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S):      BioteCon Gesellschaft fur biotechnologische Entwicklung
                         und consulting mbH, Berlin, DE, 10589 (non-U.S.
                         corporation)

                              NUMBER        KIND    DATE
                         --------------- ------ --------
PATENT INFORMATION:      US 2003059912      A1   20030327
APPLICATION INFO.:       US 2002-64903      A1   20020827  (10)
RELATED APPLN. INFO.:    Continuation-in-part of Ser. No. US 2001-700540, filed
                         on 19 Jan 2001, PENDING A 371 of International Ser. No.
```

WO 1999-EP3272, filed on 12 May 1999, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	DE 1998-19821285	19980513
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	GUDRUN E. HUCKETT, LONSSTR. 53, WUPPERTAL, 42289	
NUMBER OF CLAIMS:	11	
EXEMPLARY CLAIM:	1	
LINE COUNT:	576	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS'
ENTERED AT 11:08:30 ON 01 DEC 2003

```
L1             1 S CONJUGATE AND MASTOCYTE BINDING
L2        171149 S HYBRID PROTEIN OR CONJUGATE
L3            21 S IGE AND IGA PROTEASE
L4          1711 S IGE AND TETANUS
L5             7 S L2 AND L3
L6             0 S L4 AND MASTOCYTE INACTIVATION
L7             0 S L4 AND DEGRANULATION INHIBITION
L8          1322 S MAST CELL DEGRANULATION AND INHIBITION
L9            38 S ALLERGY AND TREATEMENT
L10            0 S L9 AND L8
L11          107 S L8 AND ALLERGIC RESPONSE
L12           88 S L11 AND IGE
L13            2 S L12 AND TETANUS TOXIN
L14            1 S LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN
L15            0 S LIGHT CHAIN TETANUS TOXIN
L16            0 S IGA PROTEASE NEISSERIA GONORRHEAE
L17         1711 S TETANUS AND IGE
L18           12 S L8 AND L17
L19          443 S CLOSTRIDIUM BOTULINUM TOXIN
L20            1 S L19 AND FC FRAGMENT
L21            2 S L19 AND L8
L22          419 S MAST CELL DEGRANULATING PEPTIDE
L23            0 S L9 AND L22
L24           19 S L22 AND IGE
L25            1 S L22 AND L19
```

=> s tetanus toxin
L26 8195 TETANUS TOXIN

=> s l26 and l22
L27 4 L26 AND L22

=> d l27 ti abs ibib tot

L27 ANSWER 1 OF 4 MEDLINE on STN

TI Neurotoxicity of apamin and MCD peptide upon central application.

AB Besides apamin, the structurally related MCD peptide (**mast cell degranulating peptide**; peptide 401) is another centrally acting peptide from bee venom. In contrast to apamin, it is hardly neurotoxic upon intravenous injection in mice. Following intraventricular injection, as little as 0.3 microgram/animal produce convulsions and respiratory arrest in mice. The clinical picture differs from that elicited by apamin, and apamin is about 10 times more potent than MCD peptide when given intraventricularly. Apamin and MCD peptide

injected into the spinal cord of rats in nanogram amounts, produce circumscript hyperexcitation lasting more than one day, however with complete recovery following sublethal doses. Local apamin poisoning differs from local tetanus (elicited by the same way) by its faster time course.

ACCESSION NUMBER: 78071874 MEDLINE
DOCUMENT NUMBER: 78071874 PubMed ID: 593441
TITLE: Neurotoxicity of apamin and MCD peptide upon central application.
AUTHOR: Habermann E
SOURCE: NAUNYN-SCHMIEDEBERGS ARCHIVES OF PHARMACOLOGY, (1977 Nov 10) 300 (2) 189-91.
Journal code: 0326264. ISSN: 0028-1298.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197802
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19970203
Entered Medline: 19780218

L27 ANSWER 2 OF 4 USPATFULL on STN
TI Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof
AB A hybrid protein contains a protein that binds to a receptor of mastocytes and basophils and is endocyted by them. The protein can be IgE; IgE fragment; IgE Fc fragment; antibody against IgE receptor of mastocytes and basophils; fragment of the antibody against the IgE receptor of mastocytes and basophils; antibody against mastocyte specific potassium channel; and **mast cell degranulating peptide**. The hybrid protein also contains a protease cleaving proteins of the secretion process of the mastocytes and basophils so as to inhibit the secretion process without killing the mastocytes and basophils. The protease can be light chain Clostridium botulinum toxin; proteolytically active fragment of the light chain of a Clostridium botulinum toxin containing an amino acid sequence His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His wherein Xaa is an amino acid; light chain of the **tetanus toxin**; proteolytically active fragment of the light chain of the **tetanus toxin** containing His-Asp-Leu-Ile-His-Val-Leu-His; IgA protease of Neisseria gonorrhoeae; and proteolytic domain of the IgA protease of Neisseria gonorrhoeae.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:86306 USPATFULL
TITLE: Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof
INVENTOR(S): Bigalke, Hans, Hannover, GERMANY, FEDERAL REPUBLIC OF
Frevert, Jurgen, Berlin, GERMANY, FEDERAL REPUBLIC OF
PATENT ASSIGNEE(S): BioteCon Gesellschaft fur biotechnologische Entwicklung und consulting mbH, Berlin, DE, 10589 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059912	A1	20030327
APPLICATION INFO.:	US 2002-64903	A1	20020827 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-700540, filed on 19 Jan 2001, PENDING A 371 of International Ser. No. WO 1999-EP3272, filed on 12 May 1999, UNKNOWN

NUMBER	DATE

PRIORITY INFORMATION: DE 1998-19821285 19980513
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GUDRUN E. HUCKETT, LONSSTR. 53, WUPPERTAL, 42289
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
LINE COUNT: 576
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L27 ANSWER 3 OF 4 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Effects of ion channel toxins and specific neurotoxins on the cyclic nucleotide content of cerebellar slices, primary brain cultures and neural cell lines.

AB cAMP and cGMP were measured in mouse cerebellar slices, neural cell lines and primary nerve cell cultures from rats after treatment with different neurotoxins and high potassium. Sea anemone toxin II (ATX II), which is known to keep the activated sodium channels open, raised the cGMP content of mouse cerebellar slices up to 35-fold and doubled their cAMP content. **Mast-cell-degranulating peptide** (MCD-peptide) from bee venom increased cGMP levels up to 15-fold. The effects of both toxins on the cyclic nucleotide content were mimicked by depolarizing agents, like high potassium and veratridine. Primary nerve cell cultures (4 weeks old) responded to ATX II and high potassium with an increase of both cGMP and cAMP, however to a smaller extent as compared with slices. Excitable structures appear to be relevant, because younger cultures (2 weeks and less) and several neural cell lines did not respond to ATX II. Specific neurotoxins like **tetanus toxin**, botulinum A toxin and apamin from bee venom had no effect on the cyclic nucleotide content of cerebellar slices and of primary nerve cell cultures. In cerebellar slices the potassium-stimulated increase of cAMP and cGMP was not affected by previous exposure of the slices to **tetanus toxin** or apamin. We conclude that opening of sodium channels in excitable membranes generally raises the cyclic nucleotide content whereas the mode of action of specific neurotoxins is not reflected by changes in the overall content of cyclic nucleotides.

ACCESSION NUMBER: 80039979 EMBASE
DOCUMENT NUMBER: 1980039979
TITLE: Effects of ion channel toxins and specific neurotoxins on the cyclic nucleotide content of cerebellar slices, primary brain cultures and neural cell lines.
AUTHOR: Ahnert G.; Glossmann H.; Habermann E.
CORPORATE SOURCE: Pharmakol. Inst., Justus Liebig-Univ. Giessen, D-6300 Lahn-Giessen 1, Germany
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology, (1979) 307/2 (151-157).
CODEN: NSAPCC
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
008 Neurology and Neurosurgery
LANGUAGE: English

L27 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN

TI EFFECTS OF ION CHANNEL TOXINS AND SPECIFIC NEURO TOXINS ON THE CYCLIC NUCLEOTIDE CONTENT OF CEREBELLAR SLICES PRIMARY BRAIN CULTURES AND NEURAL CELL LINES.

AB c[cyclic]AMP and cGMP were measured in mouse cerebellar slices, neural cell lines and primary nerve cell cultures from rats after treatment with different neurotoxins and high K. The lines used were mouse C6 glioma, mouse 108CC15 neuroblastoma .times. rat glioma hybrid and mouse Neuro 2a neuroblastoma cells. Sea anemone toxin II (ATX II) which keeps the activated Na channels open raised the cGMP content of mouse cerebellar

slices up to 35-fold and doubled their cAMP content. **Mast cell degranulating peptide** (MCD-peptide) from bee venom increased cGMP levels up to 15-fold. The effects of both toxins on the cyclic nucleotide content were mimicked by depolarizing agents, i.e., high K and veratridine. Primary nerve cell cultures (4 wk old) responded to ATX II and high K with an increase of both cGMP and cAMP but to a smaller extent as compared with slices. Excitable structures appear to be relevant because younger cultures (2 wk and less) and several neural cell lines did not respond to ATX II. Specific neurotoxins like **tetanus toxin**, botulinum A toxin and apamin from bee venom had no effect on the cyclic nucleotide content of cerebellar slices and of primary nerve cell cultures. In cerebellar slices the K-stimulated increase of cAMP and cGMP was not affected by previous exposure of the slices to **tetanus toxin** or apamin. Apparently opening of Na channels in excitable membranes generally raises the cyclic nucleotide content but the mode of action of specific neurotoxins is not reflected by changes in the overall content of cyclic nucleotide.

ACCESSION NUMBER: 1980:131595 BIOSIS
DOCUMENT NUMBER: PREV198069006591; BA69:6591
TITLE: EFFECTS OF ION CHANNEL TOXINS AND SPECIFIC NEURO TOXINS ON THE CYCLIC NUCLEOTIDE CONTENT OF CEREBELLAR SLICES PRIMARY BRAIN CULTURES AND NEURAL CELL LINES.
AUTHOR(S): AHNERT G [Reprint author]; GLOSSMANN H; HABERMANN E
CORPORATE SOURCE: PHARMAKOL INST, JUSTUS LIEBIG-UNIV GIESSEN, FRANKFURTERSTR 107, D-6300 LAHN-GIESSEN 1, W GER
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology, (1979) Vol. 307, No. 2, pp. 151-158.
CODEN: NSAPCC. ISSN: 0028-1298.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

=> d his

(FILE 'HOME' ENTERED AT 11:08:04 ON 01 DEC 2003)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, BIOSIS, BIOBUSINESS' ENTERED AT 11:08:30 ON 01 DEC 2003

L1	1	S	CONJUGATE AND MASTOCYTE BINDING
L2	171149	S	HYBRID PROTEIN OR CONJUGATE
L3	21	S	IGE AND IGA PROTEASE
L4	1711	S	IGE AND TETANUS
L5	7	S	L2 AND L3
L6	0	S	L4 AND MASTOCYTE INACTIVATION
L7	0	S	L4 AND DEGRANULATION INHIBITION
L8	1322	S	MAST CELL DEGRANULATION AND INHIBITION
L9	38	S	ALLERGY AND TREATEMENT
L10	0	S	L9 AND L8
L11	107	S	L8 AND ALLERGIC RESPONSE
L12	88	S	L11 AND IGE
L13	2	S	L12 AND TETANUS TOXIN
L14	1	S	LIGHT CHAIN CLOSTRIDIUM BOTULINUM TOXIN
L15	0	S	LIGHT CHAIN TETANUS TOXIN
L16	0	S	IGA PROTEASE NEISSERIA GONORRHEAE
L17	1711	S	TETANUS AND IGE
L18	12	S	L8 AND L17
L19	443	S	CLOSTRIDIUM BOTULINUM TOXIN
L20	1	S	L19 AND FC FRAGMENT
L21	2	S	L19 AND L8
L22	419	S	MAST CELL DEGRANULATING PEPTIDE
L23	0	S	L9 AND L22
L24	19	S	L22 AND IGE
L25	1	S	L22 AND L19

L26 8195 S TETANUS TOXIN
L27 4 S L26 AND L22

=> s l8 and l22
L28 2 L8 AND L22

=> d l28 ti abs ibib tot

L28 ANSWER 1 OF 2 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI **Inhibition** of nociceptin on sensory neuropeptide release and mast cell-mediated plasma extravasation in rats.

AB Nociceptin (20 .mu.g/kg i.p.) strongly inhibited cutaneous Evans blue accumulation in the chronically denervated hindpaw of the rat in response to **mast cell degranulating peptide** (MCDP, 0.25 .mu.g in 100 .mu.l) but it had no and marginal effect on plasma extravasation induced by 5-hydroxytryptamine (5-HT, 0.5 .mu.g in 100 .mu.l) and histamine (0.1 .mu.g in 100 .mu.l), respectively. Release of sensory neuropeptides such as substance P, calcitonin gene-related peptide (CGRP) and somatostatin from the rat isolated trachea in response to capsaicin (10-8 M) or bradykinin (10-7 M) were also attenuated by nociceptin (100 and 300 nM). It is concluded that chemically induced discharge of mediators from mast cells and from capsaicin-sensitive afferent nerve terminals are both inhibited by nociceptin that participates in the anti-inflammatory effect of the peptide.

ACCESSION NUMBER: 1998173777 EMBASE
TITLE: **Inhibition** of nociceptin on sensory neuropeptide release and mast cell-mediated plasma extravasation in rats.
AUTHOR: Nemeth J.; Helyes Z.; Oroszi G.; Than M.; Pinter E.; Szolcsanyi J.
CORPORATE SOURCE: J. Szolcsanyi, Dept. of Pharmacol./Pharmacotherapy, University Medical School of Pecs, Hungarian Academy of Sciences, P.O. Box 99, H-7643 Pecs, Hungary. szolcs@apacs.pote.hu
SOURCE: European Journal of Pharmacology, (17 Apr 1998) 347/1 (101-104).
Refs: 20
ISSN: 0014-2999 CODEN: EJPHAZ
PUBLISHER IDENT.: S 0014-2999(98)00216-7
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
LANGUAGE: English
SUMMARY LANGUAGE: English

L28 ANSWER 2 OF 2 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Nitric oxide inhibits numerous features of mast cell-induced inflammation.

AB Background: We previously reported that **mast cell degranulation** causes histamine and P-selectin dependent leukocyte rolling and platelet-activating factor (PAF)- and CD18-associated leukocyte adhesion, whereas others have reported serotonin-induced edema formation. The purpose of the present study was to determine whether nitric oxide (NO) could inhibit the mast cell- induced multistep recruitment of leukocytes and the associated microvascular dysfunction in single inflamed venules. Methods and Results: Intravital fluorescence microscopy was used to demonstrate increased leukocyte rolling and adhesion and increased albumin extravasation in single 25- to 40-.mu.m venules that were treated with the mast cell-degranulating agent compound 48/80 (CMP 48/80). The mast cell-induced histamine-dependent rolling and PAF- dependent adhesion were completely inhibited by the addition of the NO donor spermine NO. However, spermine NO did not directly inhibit histamine-induced leukocyte rolling and only partly affected PAF-induced

leukocyte adhesion. Compound 48/80- activated mast cells evoked a significant increase in PAF- dependent neutrophil adhesion in vitro. Spermine-NO prevented the mast cell- dependent neutrophil adhesion but failed to affect direct adhesion with PAF. The mast cell-induced albumin leakage was also inhibited by the NO donor. Conclusions: Taken together, these results suggest that exogenous NO can modulate leukocyte recruitment and microvascular permeability alterations elicited by mast cell activation and raises the possibility that the use of NO donors may be a reasonable therapeutic approach to reducing mast cell- dependent inflammation.

ACCESSION NUMBER: 96030819 EMBASE
DOCUMENT NUMBER: 1996030819
TITLE: Nitric oxide inhibits numerous features of mast cell-induced inflammation.
AUTHOR: Gaboury J.P.; Niu X.-F.; Kubes P.
CORPORATE SOURCE: Department of Medical Physiology, Faculty of Medicine, University of Calgary,Calgary, Alta. T2N 4N1, Canada
SOURCE: Circulation, (1996) 93/2 (318-326).
ISSN: 0009-7322 CODEN: CIRCAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
025 Hematology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English